
MEDICO-CHIRURGICAL SOCIETY
EDINBURGH

THE TRANSACTIONS

OF THE

MEDICO-CHIRURGICAL SOCIETY OF EDINBURGH.

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OF THE

MEDICO-CHIRURGICAL SOCIETY OF EDINBURGH.

VOL. VI.—NEW SERIES.

SESSION 1886-87.

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PREFACE.

The present Volume is the *Sixth* of the *New Series*, and contains a record of the work done during the Session 1886–87.

That work, as hitherto, embraces the communication of Original Papers; the exhibition of Patients, illustrating rare and interesting forms of Disease; and the exhibition of Pathological and other specimens, so essential to the proper understanding of the morbid changes which take place in the human body.

During the past Session several Extra Meetings were held for Clinical and Pathological Demonstrations, and it is hoped that such Meetings will materially increase the usefulness of the Society.

There were also held two Special Meetings, to consider and discuss the Reports of the Special Committee appointed by the Society to investigate the nature of the Contagium of Scarlet Fever. It is hoped that these reports and discussions have contributed somewhat to the elucidation of this important subject.

The Editor regrets much the great delay which has been experienced in regard to the publication of this Volume of the Transactions. The delay was caused by circumstances over which he had no control. It was partly due to Professor Fraser's valuable communication having first been published in America, and partly also to the fact that an Extra Special Meeting was held so late as October, to discuss the Report of the Committee on the Contagium of Scarlet Fever.

It is believed that the publication of the Transations in this permanent form will prove a valuable contribution to medical literature, will encourage the members to take a more active part in the work of the Society, and will tend in no small degree to increase the influence and usefulness of the Medico-Chirurgical Society of Edinburgh.

William Craig,
Editor.

April 1888.

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	John Archibald, M.D., F.R.C.S. Ed., ...	1882
130	* James Maxwell Ross, M.B., F.R.C.S. Ed., 112 Gilmore Place,	1882
	John Carlyle Johnstone, M.B., C.M., Melrose Asylum, .	1882
	James Rutherford Morison, M.D., F.R.C.S. Ed., Hartlepool, .	1882
	Roderick Maclaren, M.D., 23 Portland Square, Carlisle, .	1882
	W. Wotherspoon Ireland, M.D., Prestonpans, . . .	1883
135	Francis Mitchell Caird, M.B., F.R.C.S. Ed., 21 Rutland Street,	1883
	F. W. Dyce Fraser, M.D., F.R.C.P. Ed., South Lodge, Ascot, Berks, ...	1883
	Robert Henry Blaikie, M.D., F.R.C.S. Ed., 9 Palmerston Road,	1883
	R. M'Kenzie Johnston, M.D., F.R.C.S. Ed., 5 Rutland Square,	1883
	Charles Walker Cathcart, M.B., F.R.C.S. Ed., 8 Randolph Crescent, *Secretary*, ...	1883
140	Alexander Bruce, M.D., F.R.C.P. Ed., 13 Alva Street, .	1883
	Andrew Semple, M.D., F.R.C.S. Ed., Dep. Surgeon-General, 10 Forres Street, ...	1883
	William Hy. Shirreff, M.B., C.M., ...	1883
	John Lyon Wilson, L.R.C.P. Ed., 4 Buccleuch Place, .	1883
	Donald MacRaild, F.R.C.S. Ed., Greenock, . . .	1883
145	Henry Newcombe, M.D., F.R.C.S. Ed., 5 Dalrymple Crescent,	1883
	* Francis Troup, M.D., M.R.C.P. Ed., 1 Minto Street, . .	1883
	Russell Eliott Wood, M.B., F.R.C.S. Ed., 9 Darnaway Street,	1883
	John Macdonald Brown, M.B., F.R.C.S. Ed., 6 Atholl Place,	1883

		Date of Admission.
	James William Beeman Hodsdon, M.D., F.R.C.S. Ed., 30 Walker Street,	1883
150	John Haddon, M.D., C.M., Honeyburn, Hawick,	1883
	Germon Sims Woodhead, M.D., F.R.C.P. Ed., 6 Marchhall Crescent,	1883
	Thomas Francis Spittal Caverhill, M.B., F.R.C.P. Ed., 8A Abercromby Place,	1883
	Robert Alexander Lundie, M.B., B.Sc., F.R.C.S. Ed., 35 Warrender Park Road,	1883
	Arthur W. Hare, M.B., F.R.C.S. Ed., M.R.C.S. Eng., 21 Ainslie Place,	1883
155	Edwin Baily, M.B., C.M., Oban,	1883
	Alexander Black, M.B., F.R.C.P. Ed., 8 St Vincent Street,	1883
	Harry Melville Dunlop, M.D., F.R.C.P. Ed., 20 Abercromby Place,	1883
	George Andreas Berry, M.B., F.R.C.S. Ed., 23 Rutland Street,	1883
	Hamilton Wylie, M.B., C.M., 1 George Place,	1883
160	Arthur Donglas Webster, M.D., M.R.C.P. Ed., 20 Newington Road,	1883
	Robert William Philip, M.D., M.R.C.P. Ed., 12 Hope Street,	1883
	Joseph Carne Ross, M.D., F.R.C.P. Ed., Penzance,	1884
	William Russell, M.D., F.R.C.P. Ed., 46 Albany Street,	1884
	George Dickson, M.D., F.R.C.S. Ed., 9 India Street,	1884
165	Thomas Wyld Pairman, L.R.C.P. & S. Ed., Te Awamutu, Waipa, Auckland, N.Z.	1884
	Alexander Thom, jr., M.D., C.M., Crieff,	1884
	Hugh Logan Calder, M.D., C.M., 42 Leith Walk,	1884
	James Craig Balfour, L.R.C.P. & S. Ed., Redbourne, Kirkton-Lindsay, Lincolnshire,	1884
	Frederick Anastasius Saunders, L.R.C.P. & S. Ed., Denburn, Crail,	1884
170	William Richardson, M.D., F.R.C.S. Ed., Bath Lodge, Reading,	1884
	Andrew Brown, M.D., M.R.C.P. Ed., 1 Bartholomew Road, Kentish Town, London, N.W.	1884
	G. J. H. Bell, M.B., C.M., Surgeon, Bengal Army,	1884
	T. Goodall Nasmyth, M.B., C.M., Cowdenbeath, Fife,	1884
	Henry Hay, M.B., C.M., 7 Brandon Street,	1884
175	Thomas R. Scott, M.D., C.M., Musselburgh,	1884
	R. Milne Murray, M.B., F.R.C.P. Ed., 10 Hope Street,	1884
	A. Murray Gibson, M.D., Portobello,	1884
	A. S. Cumming, M.D., F.R.C.P. Ed., 18 Ainslie Place,	1884
	Ernest F. Neve, M.D., F.R.C.S. Ed., M.R.C.S. Eng., Hospital, Srinagar, Kashmir, N.W. India,	1884
180	W. C. Greig, M.B., C.M., 69 Church Street, St Helens, Lancashire,	1884
	William Wilson, M.B., C.M., 21 Young Street,	1885
	John Mowat, M.D., 1 Hope Park Terrace,	1885
	Skene Keith, M.B., F.R.C.S. Ed., 42 Charles Street, Berkeley Square, London, W.,	1885
	D. Noël Paton, M.D., F.R.C.P. Ed., 4 Walker Street,	1885
185	George Hugh Mackay, M.B., C.M., Elgin,	1885
	J. Michael Dewar, M.B., C.M., 110 Lauriston Place,	1885
	Edward M'Callum, F.R.C.S. Ed., 3 Brandon Street,	1885
	T. Edgar Underhill, M.D., F.R.C.S. Ed., Bromsgrove, Worcestershire,	1885
	John Struthers Stewart, L.R.C.P. & S. Ed., 16 Merchiston Terrace,	1885
190	Allen Thomson Sloan, M.D., C.M., 22 Forth Street,	1885
	John William Ballantyne, M.B., C.M., 50 Queen Street,	1885
	James Robertson Crease, F.R.C.S. Ed., 2 Ogle Terrace, South Shields,	1885

		Date of Admission.
	George Kerr, M.B., C.M., 9 Great Stuart Street,	1885
	Tom Bairstow, L.R.C.P. & S. Ed., 13 Buccleuch Place,	1885
195	David Milligan, M.B., C.M., 7 West Maitland Street,	1885
	George Dods, M.D., L.R.C.S. Ed., 36 Moray Place,	1885
	J. Murdoch Brown, M.B., F.R.C.P. Ed., 9 Walker Street,	1885
	Robert W. Felkin, M.D., F.R.C.S. Ed., 20 Alva Street,	1885
	S. Hale Puckle, M.B., C.M., Bishop Castle, Shropshire,	1885
200	James Haig Ferguson, M.B., M.R.C.P. Ed., M.R.C.S. Eng., 16 Hope Street,	1885
	Charles Kennedy, M.D., C.M., 25 Newington Road,	1886
	William Gayton, M.D., M.R.C.S. Eng., Bartram Lodge, Fleet Road, Hampstead, London, N.W.,	1886
	Reginald Ernest Horsley, M.B., C.M. (Communications to Messrs E. & S. Livingstone, 15 Teviot Place),	1886
	James Mill, M.B., C.M., 178 Ferry Road,	1886
205	Robert Fraser Calder Leith, M.B., B.Sc., 107 Marchmont Road,	1886
	Thomas M. Burn-Murdoch, M.B., C.M., 31 Morningside Road,	1886
	Professor William Smith Greenfield, M.D., F.R.C.P. Lond. and Ed., 7 Heriot Row,	1886
	Oswald Gillespie Wood, M.D., F.R.C.S. Ed., Surgeon, Army Medical Staff, The Castle,	1886
	James Hogarth Pringle, M.B., C.M., 5 Livingstone Place.	1886
210	Nathaniel Thomas Brewis, M.B., F.R.C.P. Ed., 59 Queen Street,	1886
	John Batty Tuke, jr., M.B., M.R.C.P. Ed., Balgreen, Murrayfield,	1886
	David Berry Hart, M.D., F.R.C.P. Ed., 4 Wemyss Place,	1886
	Walter Scott Lang, M.D., F.R.C.S. Ed., M.R.C.S. Eng., 1 Leopold Place,	1886
	Alfred Bell Whitton, M.B., C.M., Aberchirder,	1886
215	Robert S. Aitchison, M.B., C.M., 74 Great King Street,	1887
	J. A. Armitage, M.B., C.M., 15 Waterloo Road, Wolverhampton,	1887
	J. Walton Hamp, L.F.P.S. Glasg., L.S.A. Lond., Wolverhampton,	1887
	William Hunter, M.D., M.R.C.S. Eng., The University, Cambridge,	1887
	Sydney Rumboll, L.R.C.P. and S. Ed., Grangemouth,	1887
220	John Thomson, M.B., C.M., 14 Coates Crescent,	1887
	George Franklin Shiels, M.B., C.M., 3 Greenhill Place,	1887
	T. Brown Darling, M.B., C.M., 36 South Bruntsfield Place,	1887
	John Keay, M.B., C.M., Mavisbank House, Polton,	1887
	John F. Sturrock, M.B., C.M., Golspie,	1887
225	Edward Carmichael, M.D., 8 Mansfield Place,	1887
	Charles C. Teacher, M.B., C.M., 5 Newington Road,	1887
	David W. Aitken, M.B., C.M., 3 Argyle Place,	1887
	Robert Inch, M.B., C.M., Gorebridge,	1887
	John Shaw M'Laren, M.B., C.M., 14 Walker Street,	1887
230	George Mackay, M.B., F.R.C.S. Ed., M.R.C.S. Eng., 2 Randolph Place,	1887
	Henry Alexis Thomson, M.B., C.M., 6A Bruntsfield Place,	1887
	David Wallace, M.B., C.M., 66 Northumberland Street,	1887
	John C. Messer, M.D., R.N., 1 Lansdowne Crescent,	1887

NON-RESIDENT.

	Arthur Edward Turnour, M.D., M.R.C.S. Eng., *Denbigh*,	1843
235	W. Ord M'Kenzie, M.D., L.R.C.S. Ed., *London*,	1845
	W. Judson Van Someren, M.D., L.R.C.S. Ed., *Redhill, Surrey*,	1845
	William H. Lowe, M.D., F.R.C.P. Ed., *Wimbledon*,	1845
	George Skene Keith, M.D., F.R.C.P. Ed., *Currie*,	1845
	W. Chalmers Fowler, M.D., M.R.C.S. Eng., *London*,	1847
240	Veitch Sinclair, L.R.C.P. and S. Ed., *London*,	1850
	Andrew Graham, M.D., Fleet Surgeon, R.N.,	1853

		Date of Admission.
	Archibald Hall, M.D., *Montreal*,	1853
	John Traill, F.R.C.S. Ed., *Arbroath*,	1853
	W. Overend Priestly, M.D., LL.D., F.R.C.P. Ed., *London*,	1854
245	Horatio Robinson Storer, M.D., *Newport, Rhode Island, U.S.*,	1855
	James C. Howden, M.D., *Montrose*,	1856
	Thomas Skinner, M.D., L.R.C.S. Ed., *London*,	1856
	Professor William Smoult Playfair, M.D., F.R.C.P.L., *London*,	1857
	J. Ivor Murray, M.D., F.R.C.S. Ed., *Scarboro'*,	1857
250	Andrew Scott Myrtle, M.D., L.R.C.S. Ed., *Harrogate*,	1859
	Robert Foulis, M.D., F.R.C.S. Ed., *Cupar-Fife*,	1859
	Francis Robertson Macdonald, M.D., *Inveraray*,	1860
	Professor John Young, M.D., *University of Glasgow*,	1860
	Norman Bethune, M.D., F.R.C.S. Ed., *Toronto*,	1861
255	George Thin, M.D., L.R.C.S. Ed., *London*,	1861
	Peter Gordon, L.R.C.P. and S. Ed., *Juniper Green*,	1861
	J. Cecil Phillippo, M.D., *Kingston, Jamaica*,	1861
	Professor William Stephenson, M.D., F.R.C.S. Ed., *Aberdeen*,	1861
	David Yellowlees, M.D., F.F.P.S. Glasg., *Glasgow*,	1862
260	William M'Culloch Watson, M.D., *Montrose*,	1863
	Prof. Arthur Gamgee, M.D., F.R.C.P. Ed., F.R.S., *St Leonards-on-Sea*,	1864
	Professor John Cleland, M.D., LL.D., *The University, Glasgow*,	1864
	R. B. Finlay, M.D., M.P., *Middle Temple, London*,	1864
	Stanley Lewis Haynes, M.D., M.R.C.S. Eng., *Malvern*,	1864
265	Francis D. A. Skae, M.D., *Lerwick*,	1864
	James Watt Black, M.D., F.R.C.P.L., *London*,	1865
	David Brodie, M.D., *Canterbury*,	1865
	Thomas Sheriff, L.R.C.P. and S. Ed., *Edinburgh*,	1867
	Peter Maury Deas, M.B., L.R.C.S. Ed., *Exeter*,	1868
270	Professor J. G. M'Kendrick, M.D., F.R.C.P. Ed., *University, Glasgow*,	1870
	Lawson Tait, M.D., F.R.C.S. Ed. and Eng., *Birmingham*,	1870
	J. G. Sinclair Coghill, M.D., F.R.C.P. Ed., *Ventnor*,	1870
	James Johnston, M.D., L.R.C.S. Ed., *Shanghai*,	1871
	J. William Eastwood, M.D., M.R.C.P.L., *Darlington*.	1871
275	Professor J. Bell Pettigrew, M.D., LL.D., F.R.C.P. Ed., *University of St Andrews*,	1873
	John Smith,	1873
	John Aymers Macdougall, M.D., F.R.C.S. Ed., *Carlisle*,	1875
	Thomas John Maclagan, M.D., M.R.C.P.L., *London*,	1875
	Dr Groesbeck, *Cincinnati*,	1875
280	Professor David James Hamilton, M.B., F.R.C.S. Ed., *Aberdeen University*,	1876
	J. Moolman, M.B., C.M., *Cape of Good Hope*,	1877
	Robert Somerville, M.D., L.R.C.S. Ed., *Galashiels*,	1877
	Graham Steell, M.D., M.R.C.P.L., *Manchester*,	1877
	Frederick William Barry, M.D., D.Sc., *London*,	1878
285	Thomas Inglis, F.R.C.P. Ed., *Lincoln*,	1878
	John Brown, M.D., F.R.C.S. Eng., *Burnley*,	1878
	Walter Weir, M.B., F.R.C.P. Ed., *London*,	1879
	Keith Norman Macdonald, M.D., F.R.C.P. Ed., *Cupar-Fife*,	1880
	John Home Hay, M.D., M.R.C.S. Eng., *Alloa*,	1880
290	John Mackay, M.D., L.R.C.S. Ed., *Aberfeldy*,	1881

ORDINARY MEMBERS

ARRANGED ALPHABETICALLY.

RESIDENT.

Dr J. O. Affleck, 38 Heriot Row,	1871
Dr R. S. Aitchison, 74 Great King Street,	1887

		Date of Admission.
	Dr D. Aitken, 3 Argyle Place,	1887
	Dr James Andrew, 2 Atholl Crescent,	1869
5	Professor Annandale, 34 Charlotte Square,	1863
	Dr Archibald,	1882
	Dr J. A. Armitage, 15 Waterloo Road, Wolverhampton,	1887
	Dr W. Badger, Penicuik,	1882
	Dr J. Johnson Bailey, Marple, Cheshire,	1874
10	Dr Edwin Baily, Oban,	1883
	Tom Bairstow, Esq., 13 Buccleuch Place,	1885
	Dr Andrew Balfour, Portobello,	1874
	Dr J. H. Balfour, Portobello,	1881
	Dr G. W. Balfour, 17 Walker Street,	1874
15	Dr James Craig Balfour, Redbourne, Kirkton-Lindsay, Lincolnshire,	1884
	Dr Thomas Balfour, 51 George Square,	1856
	Dr Alexander Ballantyne, Dalkeith,	1872
	Dr J. W. Ballantyne, 50 Queen Street,	1885
	Dr A. H. Freeland Barbour, 24 Melville Street,	1881
20	Dr G. J. H. Bell, Surgeon, Bengal Army,	1884
	Joseph Bell, Esq., 2 Melville Crescent,	1862
	G. H. Bentley, Esq., Kirkliston,	1877
	Dr G. A. Berry, 23 Rutland Street,	1883
	Dr James S. Beveridge, 8 Eildon Street,	1861
25	Dr Alexander Black, 8 St Vincent Street,	1883
	Dr W. T. Black, 2 George Square,	1877
	Dr Robert H. Blaikie, 9 Palmerston Road,	1883
	Dr Bleloch, 2 Lonsdale Terrace,	1871
	Dr Brakenridge, 10 St Colme Street,	1865
30	Dr Byrom Bramwell, 23 Drumsheugh Gardens,	1876
	Dr N. T. Brewis, 59 Queen Street,	1886
	Dr Brown, 1 Bartholomew Road, Kentish Town, London, N.W.,	1884
	Dr J. Graham Brown, 16 Ainslie Place,	1878
	Dr J. Macdonald Brown, 6 Atholl Place,	1883
35	Dr J. Murdoch Brown, 9 Walker Street,	1885
	Dr Alexander Bruce, 13 Alva Street,	1883
	Dr Robert Bruce, 12 York Place,	1858
	Dr Buist, 1 Clifton Terrace,	1877
	Dr T. M. Burn-Murdoch, 31 Morningside Road,	1886
40	Dr Cadell, 5 Castle Terrace,	1870
	Dr Francis M. Caird, 21 Rutland Street,	1883
	Dr H. L. Calder, 42 Leith Walk,	1884
	Dr W. Watson Campbell, Duns,	1877
	Dr Cappie, 47 Lauriston Place,	1855
45	Dr Edward Carmichael, 8 Mansfield Place,	1887
	Dr J. Carmichael, 22 Northumberland Street,	1870
	Dr C. W. Cathcart, 8 Randolph Crescent, *Secretary*,	1883
	Dr T. F. S. Caverhill, 8A Abercromby Place,	1884
	Professor John Chiene, 26 Charlotte Square, *Vice-President*,	1867
50	Dr Church, 36 George Square,	1876
	Dr Clouston, Tipperlinn House, Morningside Place,	1861
	Dr A. R. Coldstream, Florence, Italy,	1878
	Dr John Connel, Peebles,	1876
	Dr Cotterill, 23 Walker Street,	1878
55	Dr William Craig, 7 Bruntsfield Place,	1869
	Dr J. R. Crease, 2 Ogle Terrace, South Shields,	1885
	Dr Halliday Croom, 25 Charlotte Square,	1870
	Dr A. S. Cumming, 18 Ainslie Place,	1884
	Dr R. J. B. Cunynghame, 6 Walker Street,	1868
60	Dr J. B. Darling, 36 South Bruntsfield Place,	1887
	Dr J. M. Dewar, 110 Lauriston Place,	1885
	Dr Archibald Dickson, 11 Royal Circus,	1871
	Dr George Dickson, 9 India Street,	1884

		Date of Admission.
	Dr George Dods, 36 Moray Place,	1885
65	Dr Halliday Douglas, 30 Melville Street,	1842
	Dr William B. Dow, Dunfermline,	1879
	Dr John Duncan, 8 Ainslie Place, *Vice-President*,	1868
	Dr Kirk Duncanson, 22 Drumshengh Gardens,	1871
	Dr H. M. Dunlop, 20 Abercromby Place,	1883
70	Dr J. Dunsmure, 53 Queen Street,	1872
	C. H. Fasson, Esq., Dep. Surg.-Gen., Royal Infirmary,	1879
	Dr R. W. Felkin, 20 Alva Street,	1885
	Dr J. Haig Ferguson, 16 Hope Street,	1885
	Dr W. A. Finlay, St Helen's, Russell Place,	1875
75	Dr Andrew Fleming, 8 Napier Road,	1880
	Dr Foulis, 34 Heriot Row,	1875
	Dr F. W. Dyce Fraser, South Lodge, Ascot, Berks,	1883
	Dr John Fraser, 19 Strathearn Road,	1878
	Professor Thomas R. Fraser, 37 Melville Street,	1865
80	Dr R. Freeland, Broxburn,	1879
	Dr Garland, 35 Charlotte Street, Leith,	1873
	Dr W. Gayton, Bartram Lodge, Fleet Road, Hampstead, London, N.W.,	1886
	Dr A. Murray Gibson, Portobello,	1884
	Dr G. A. Gibson, 17 Alva Street,	1880
85	Dr James D. Gillespie, 10 Walker Street,	1852
	G. R. Gilruth, Esq., 67 York Place,	1869
	Dr J. Allan Gray, 107 Ferry Road,	1879
	Professor Greenfield, 7 Heriot Row,	1886
	Dr David Greig, 38 Coates Gardens,	1854
90	Dr W. C. Greig, 69 Church Street, St Helen's, Lancashire,	1884
	Dr R. H. Gunning, 12 Addison Crescent, West Kensington, London, W.,	1846
	Dr John Haddon, Honeyburn, Hawick,	1883
	Dr J. W. Hamp, Wolverhampton,	1887
	Dr A. W. Hare, 21 Ainslie Place,	1883
95	Dr D. Berry Hart, 4 Wemyss Place,	1886
	Dr Henry Hay, 7 Brandon Street,	1884
	Dr John Henderson, 7 John's Place, Leith,	1848
	Dr J. W. B. Hodsdon, 30 Walker Street,	1883
	Dr R. E. Horsley (Communications to Messrs E. & S. Livingstone, 15 Teviot Place),	1886
100	Dr George Hunter, Linlithgow,	1876
	Dr James A. Hunter, 18 Abercromby Place,	1851
	Dr W. Hunter, The University, Cambridge,	1887
	Dr Husband, 28 Clarence Street,	1849
	Francis B. Imlach, Esq., 48 Queen Street,	1843
105	Dr Robert Inch, Gorebridge,	1887
	Dr Archibald Inglis, 33 Albany Street,	1827
	Dr W. Wotherspoon Ireland, Prestonpans,	1883
	Dr James, 44 Melville Street,	1877
	Dr W. Allan Jamieson, 26 Rutland Street,	1876
110	Dr James Jamieson, 43 George Square,	1877
	Dr R. M'Kenzie Johnston, 5 Rutland Square,	1883
	Dr J. Carlyle Johnstone, Melrose Asylum,	1882
	Dr J. Keay, Mavisbank House, Polton,	1887
	Dr Keiller, 21 Queen Street,	1845
115	Dr Skene Keith, 42 Charles Street, Berkeley Square, London, W.,	1885
	Dr Thomas Keith, 42 Charles Street, Berkeley Square, London, W.,	1852
	Dr C. Kennedy, 25 Newington Road,	1886
	Dr George Kerr, 9 Great Stuart Street,	1885
	Dr W. Scott Lang, 1 Leopold Place,	1886
120	Dr Robert Lawson, 24 Mayfield Terrace,	1881

		Date of Admission.
	Dr R. F. C. Leith, 107 Marchmont Road,	1886
	Dr George Leslie, Falkirk,	1881
	Dr Linton, 60 George Square,	1863
	Dr Littlejohn, 24 Royal Circus,	1853
125	Dr Lucas, Dalkeith,	1875
	Dr R. A. Lundie, 35 Warrender Park Road,	1883
	Dr P. M'Bride, 16 Chester Street,	1879
	Dr E. M'Callum, 3 Brandon Street,	1885
	John M'Gibbon, Esq., 55 Queen Street,	1868
130	Dr MacGillivray, 11 Rutland Street,	1877
	Dr G. Mackay, 2A Gilmore Place,	1878
	Dr George Mackay, 2 Randolph Place,	1887
	Dr G. H. Mackay, Elgin,	1885
	Professor Sir Douglas Maclagan, 28 Heriot Row,	1834
135	Dr J. S. M'Laren, 14 Walker Street,	1887
	Dr P. H. Maclaren, 1 Drumsheugh Gardens,	1868
	Dr Roderick M'Laren, 23 Portland Square, Carlisle,	1882
	Dr Donald MacRaild, Greenock,	1883
	Dr A. Matthew, Corstorphine,	1882
140	Dr D. Menzies, 21 Rutland Square,	1878
	Dr W. Menzies, 115 Lothian Road,	1847
	Dr J. C. Messer, 1 Lansdowne Crescent,	1887
	Dr J. Mill, 178 Ferry Road,	1886
	A. G. Miller, Esq., 7 Coates Crescent, *Treasurer*,	1867
145	Dr D. Milligan, 7 West Maitland Street,	1885
	Sir Arthur Mitchell, 34 Drummond Place,	1859
	Dr Moir, 52 Castle Street,	1836
	Dr Alexander Moir, 30 Buccleuch Place,	1876
	Dr J. Rutherford Morison, Hartlepool,	1882
150	Dr John Mowat, 1 Hope Park Terrace,	1885
	Dr Claud Muirhead, 30 Charlotte Square,	1866
	Dr R. Milne Murray, 10 Hope Street,	1884
	Dr A. D. Leith Napier, 3 Beaufort Gardens, London,	1879
	Dr T. Goodall Nasmyth, Cowdenbeath, Fife,	1884
155	Dr E. F. Neve, Dispensary, Srinagar, Kashmir, N.W. India,	1884
	Dr H. Newcombe, 5 Dalrymple Crescent,	1883
	Dr P. Orphoot, 113 George Street,	1865
	Dr T. W. Pairman, Te Awamutu, Waipa, Auckland, N.Z.,	1884
	Dr Paterson, 4 Coates Crescent,	1847
160	Dr D. Noël Paton, 4 Walker Street,	1885
	Dr Peddie, 15 Rutland Street,	1842
	Dr J. A. Philip, Rue Victor Hugo, Boulogne-Sur-Mer,	1878
	Dr R. W. Philip, 12 Hope Street,	1883
	Dr Playfair, 25 Rutland Street,	1874
165	Dr J. H. Pringle, 5 Livingstone Place,	1886
	Dr S. Hale Puckle, Bishop Castle, Shropshire,	1885
	Dr Rattray, Portobello,	1874
	Dr William Richardson, Bath Lodge, Reading,	1884
	Dr James Ritchie, 14 Charlotte Square, *Secretary*,	1873
170	Dr R. Peel Ritchie, 1 Melville Crescent, *Vice-President*,	1862
	Dr Argyll Robertson, 18 Charlotte Square,	1861
	Dr Ronaldson, 18 Bruntsfield Place,	1877
	Dr J. Maxwell Ross, 112 Gilmore Place,	1882
	Dr Joseph C. Ross, Penzance,	1884
175	Dr S. Rumboll, Grangemouth,	1887
	Dr William Russell, 46 Albany Street,	1884
	Professor Rutherford, 14 Douglas Crescent,	1866
	Dr F. A. Saunders, Denburn, Crail,	1884
	Dr Thomas R. Scott, Musselburgh,	1884
180	Dr Andrew Semple, 10 Forres Street,	1883
	Dr John Shand, 34 Albany Street,	1878
	C. H. E. Sheaf, Esq., Toowoomba, Queensland, Australia,	1871

		Date of Admission.
	Dr G. F. Shiels, 3 Greenhill Place,	1887
	Dr W. H. Shirreff,	1883
185	Dr J. Sibbald, 3 St Margaret's Road,	1859
	Professor Simpson, 52 Queen Street,	1859
	Dr A. J. Sinclair, 21 Northumberland Street,	1873
	Dr A. T. Sloan, 22 Forth Street,	1885
	Dr Andrew Smart, 20 Charlotte Square,	1865
190	Dr G. D. Smith, 146 Ferry Road,	1877
	Dr John Smith, 11 Wemyss Place,	1856
	Professor Grainger Stewart, 19 Charlotte Square, *President*,	1861
	Dr J. S. Stewart, 16 Merchiston Terrace,	1885
	Dr W. Stewart, Kirkwall,	1879
195	Dr John Strachan, Dollar,	1867
	Dr James Struthers, 39 Charlotte Street, Leith,	1849
	Dr J. F. Sturrock, Golspie,	1887
	Dr Johnson Symington, 2 Greenhill Park,	1878
	Dr W. Taylor, 12 Melville Street,	1871
200	Dr C. C. Teacher, 5 Newington Road,	1887
	Dr C. H. Thatcher, 13 Albany Street,	1876
	Dr Alexander Thom, jr., Crieff,	1884
	Dr Alexander Thomson, 14 Rankeillor Street,	1849
	Dr H. A. Thomson, 6A Bruntsfield Place,	1887
205	Dr John Thomson, 14 Coates Crescent,	1887
	Dr J. Stitt Thomson, Dalkeith,	1877
	Dr Francis Troup, 1 Minto Street,	1883
	Dr Batty Tuke, 20 Charlotte Square,	1864
	Dr J. Batty Tuke, jr., Balgreen, Murrayfield,	1886
210	Professor Sir William Turner, 6 Eton Terrace,	1858
	Dr R. S. Turner, Keith,	1867
	Dr Underhill, 8 Coates Crescent,	1872
	Dr T. Edgar Underhill, Broomsgrove, Worcestershire,	1885
	Dr D. Wallace, 66 Northumberland Street,	1887
215	B. C. Waller, Esq., M.B., Cowen-Bridge, Kirkby-Lonsdale,	1877
	Dr P. H. Watson, 16 Charlotte Square,	1856
	Dr W. Watson, Midcalder,	1862
	Dr A. D. Webster, 20 Newington Road,	1883
	Dr Graham Weir, 36 Heriot Row,	1843
220	Dr A. B. Whitton, Aberchirder,	1886
	Dr David Wilson, 12 Dean Terrace,	1844
	J. L. Wilson, Esq., 4 Buccleuch Place,	1883
	Dr T. D. Wilson, 10 Newington Road,	1880
	Dr William Wilson, 21 Young Street,	1885
225	Dr Oswald G. Wood, The Castle,	1886
	Dr Russell E. Wood, 9 Darnaway Street,	1883
	Dr G. Sims Woodhead, 6 Marchhall Crescent,	1883
	Dr Strethill Wright, 8 St Aidan's Terrace, Claughton, Birkenhead,	1871
	Dr Hamilton Wylie, 1 George Place,	1883
230	Dr John Wyllie, 1 Melville Street,	1868
	Dr James Young, 14 Ainslie Place,	1859
	Dr P. A. Young, 25 Manor Place,	1870
	Dr Ziegler, 47 George Square,	1876

NON-RESIDENT.

	Dr F. W. Barry, *London*,	1878
235	Dr Bethune, *Toronto*,	1861
	Dr J. W. Black, *London*,	1865
	Dr Brodie, *Canterbury*,	1865

		Date of Admission.
	Dr John Brown, *Burnley*,	1878
	Professor Cleland, *Glasgow*,	1864
240	Dr Coghill, *Ventnor*,	1870
	Dr P. M. Deas, *Exeter*,	1868
	Dr J. W. Eastwood, *Darlington*,	1871
	Dr R. B. Finlay, M.P., *Middle Temple, London*,	1864
	Dr Foulis, *Cupar-Fife*,	1859
245	Dr Fowler, *London*,	1847
	Professor Gamgee, *St-Leonards-on-Sea*,	1863
	Peter Gordon, Esq., *Juniper Green*,	1861
	Dr A. Graham, R.N.,	1853
	Dr Groesbeck, *Cincinnati*,	1875
250	Dr Archibald Hall, *Montreal*,	1853
	Professor D. J. Hamilton, *Aberdeen University*,	1876
	Dr J. H. Hay, *Alloa*,	1880
	Dr Stanley Haynes, *Malvern*,	1864
	Dr J. S. Howden, *Montrose*,	1856
255	Dr T. Inglis, *Lincoln*,	1878
	Dr James Johnston, *Shanghai*,	1871
	Dr George Keith, *Currie*,	1845
	Dr Lowe, *Wimbledon*,	1845
	Dr F. R. Macdonald, *Inveraray*,	1860
260	Dr K. N. Macdonald, *Cupar-Fife*,	1880
	Dr John A. Macdougall, *Carlisle*	1875
	Dr John Mackay, *Aberfeldy*,	1881
	Professor M'Kendrick, *Glasgow*,	1870
	Dr W. O. Mackenzie, D.I.G.H., *London*,	1845
265	Dr T. J. Maclagan, *London*,	1875
	Dr J. Moolman, *Cape of Good Hope*,	1877
	Dr J. Ivor Murray, *Scarboro'*,	1857
	Dr Andrew Myrtle, *Harrogate*,	1859
	Professor Bell Pettigrew, *St Andrews*,	1873
270	Dr Phillippo, *Kingston, Jamaica*,	1860
	Professor W. S. Playfair, *London*,	1857
	Dr Priestley, *London*,	1854
	Thomas Sheriff, Esq., *Edinburgh*,	1867
	Dr Sinclair, *London*,	1850
275	Dr Francis Skae, *Lerwick*,	1864
	Dr T. Skinner, *London*,	1856
	Dr John Smith,	1873
	Dr Van Someren, *Redhill, Surrey*,	1845
	Dr Somerville, *Galashiels*,	1877
280	Dr Graham Steell, *Manchester*,	1877
	Professor Stephenson, *Aberdeen*,	1861
	Dr H. R. Storer, *Newport, Rhode Island, U.S.*,	1855
	Dr Lawson Tait, *Birmingham*,	1870
	Dr Thin, *London*,	1861
285	John Traill, Esq., *Arbroath*,	1853
	Dr Turnour, *Denbigh*,	1843
	Dr W. Watson, *Montrose*,	1863
	Dr Walter Weir, *London*,	1879
	Dr Yellowlees, *Gartnavel Asylum, Glasgow*,	1862
290	Professor John Young, *Glasgow*,	1860

N.B.—*Members are requested to communicate with the Secretaries if they discover any errors or omissions in the List, and also to intimate all changes in their addresses.*

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TRANSACTIONS

OF

THE MEDICO-CHIRURGICAL SOCIETY OF EDINBURGH,

FOR SESSION LXVI., 1886-87.

Meeting I.—November 3, 1886.

Professor GRAINGER STEWART, *President, in the Chair.*

I. ELECTION OF OFFICE-BEARERS.

THE following were elected office-bearers for Session 1886-7:—*President,* Prof. Grainger Stewart; *Vice-Presidents,* Dr John Duncan, Dr Peel Ritchie, Prof. Chiene; *Councillors,* Dr John Smith, Dr Littlejohn, Dr Troup, Dr Allan Gray, Dr James, Dr Leith Napier (Dunbar), Dr T. Duddingston Wilson, Dr G. A. Gibson; *Treasurer,* Mr A. G. Miller; *Secretaries,* Dr Cathcart, 44 Melville Street; Dr James Ritchie, 14 Charlotte Square; *Editor of Transactions,* Dr William Craig, 7 Bruntsfield Place.

II. EXHIBITION OF PATIENTS.

1. *Dr Byrom Bramwell* showed a case of HEMIPLEGIA due to an injury of the head at the time of birth.

Dr Craig asked if a depression existed now.

Dr Clouston said he had had a dozen such cases under his care, and had post-mortems in a number of them. These indicated that trephining would be quite useless. The opposite hemisphere was usually more or less undeveloped, and the cranium much thickened on that side.

Dr James asked if measurements had been taken of the boy's head. He thought it looked rather small, but it might not be so for his age.

Dr Bramwell stated in reply that there was now no apparent evidence of the former injury. The two sides of the head appeared to be symmetrical. He thought Dr James's suggestion a valuable one, and would have the head accurately measured.

2. *Dr Cotterill* showed, for Professor Annandale, a boy who had been successfully operated on for INTUSSUSCEPTION in the Sick Children's Hospital.

3. *Dr Cotterill* showed a man who had made a satisfactory recovery from severe traumatic TETANUS. He had sustained a wound of the ball of his right thumb in getting over a wall covered with broken bottles. Symptoms of tetanus having occurred, he was put on physostigma by his own medical attendant, but without any benefit. He came into hospital on the sixteenth day after the injury, and was treated for some days with chloral and bromide. Eserine (gr. $\frac{1}{13}$th) was given twice after consultation with Professor Fraser, and icebags were applied to the spine. As he did not improve, the cicatrix was freely excised. Since the operation there had been no recurrence of the fits.

III. EXHIBITION OF SPECIMENS AND PHOTOGRAPHS.

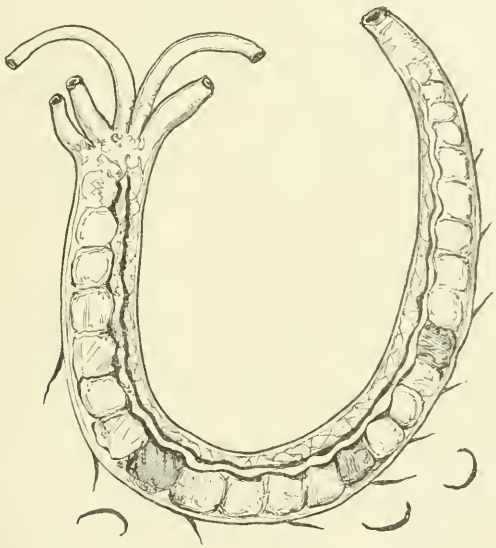
1. *Dr Byrom Bramwell* showed—(1) photographs of a CHILD affected with OPHTHALMOPLEGIA EXTERNA ACUTA, which showed the condition at the height of the paralysis and after recovery; (2) also a photograph of the HAND of the same patient, showing an enlargement of the first phalanx of the middle finger of the right hand, which apparently was the result of scrofulous disease of the bone; (3) a series of photographs illustrative of a TUMOUR of the PITUITARY BODY; (4) a large ANEURISM of the RIGHT POSTERIOR CEREBRAL ARTERY, and a series of photographs illustrative of the position of the aneurism and its relationship to the brain tissue.

2. *Mr A. G. Miller* showed—(*a*) a VESICAL CALCULUS removed by a new method devised by Professor Annandale, which consisted in gripping the stone with a lithotrite, projecting it above the pubis against the abdominal wall, and cutting down upon it. He found the operation exceedingly easy, and the result had been a success. (*b*) Parts from a case of INTUSSUSCEPTION successfully operated on. The intestine was unravelled with comparative facility, but there was bronchitis at the time of operation, from which the child ultimately died. There were no symptoms of peritonitis, and no trace of it was observed at the post-mortem. Mr Caird had made a cast showing the conditions after the operation. (*c*) Parts from a case of EXCISION of the KNEE for anchylosis. Along with the diseased parts was a piece of skin, which Mr Miller

said he was now in the habit of removing to prevent the redundancy so often seen after that operation.

3. *Dr Church* showed, in connexion with his paper on a case of poisoning by corrosive sublimate, a sample of the FLUID taken, and pieces of COPPER FOIL treated with arsenic and mercury respectively in Reinsch's tests for these substances; also the difference microscopically of sublimed globules of mercury and sublimed rounded crystals of arsenious acid.

4. *Mr Maxwell Ross* showed, for Dr Kirk Duncanson, a drawing of an ANIMAL ORGANISM removed from the ear of a native woman in India by Surgeon J. More Reid, of Karachi. It appeared along with two others out of a piece of softened wax which had been washed out of the meatus, was about a quarter of an inch in length, of the thickness of thick thread, and tapered at both ex-



The three small curves below the figure represent the natural size of the organism. The drawing was made by Dr W. Bertie Mackay.

tremities. The drawing of it was made under a low power of the microscope ($\frac{1}{4}$-inch objective). All of them were very active in their movements in a basin of water, in which they remained for four days, and were only killed in manipulating them on to a slide. She accounted for their presence by stating that similar larvæ appeared after she had slept near a heap of cow-dung on visiting her daughter at Bombay some months previous.

5. *Mr Joseph Bell* showed a large FIBROMA removed successfully from the right iliac region and pelvis of a man.

From notes by Mr John Garvie, house-surgeon.—Donald Macdonald, 48, from Knockholium, Stratherrick, Inverness-shire, shepherd, was admitted on 1st September, suffering from large tumour in groin.

Family History.—Father alive and healthy. Mother died when he was 4 years old, he thinks, of consumption. Sister died in childbirth. Has four of family, all alive and well.

Habits.—Lives in high country, and is accustomed to be out night and day in all sorts of weather. Lives in a damp house, but when tumour appeared he lived in a good house. Is not a teetotaller; is not accustomed to drink; as shepherd he had to do for long intervals without food.

Previous Health.—Never remembers a day's illness.

History of Present Attack.—In April two and a half years ago a small lump appeared in the groin (?) It slowly grew, extending in line of groin both outwards and inwards. In about six months it passed into scrotum; for about four months he has noticed no difference in size. During last winter, when it was attaining its present size, it was extending not so much outwards and inwards as projecting from the natural line of the body. He has worked up till last Saturday, and then those working with him never knew that there was anything wrong with him. He worked as well as ever he did—only now the bulk of the tumour interfered with him stooping. There are no abnormal sensations in left leg; he can move it as well as the other.

Local Condition.—A large oval projecting mass is seen in right groin, passing into and distending the scrotum, turning penis to the left side. Above it leaves the line of Poupart's ligament, lies on its abdominal side, and projects upwards into right lumbar region. It has a steep side towards the thigh, which it overhangs. On the abdominal side it falls more gradually, reaching to within $2\frac{1}{4}$ inches from the umbilicus (on the left side there is 6 inches between the umbilicus and Poupart's ligament). The tumour has a smooth surface, but can be seen to be made up of four or five large masses. On the summit there are various small reddened elevations. It measures 12 inches long, $8\frac{1}{4}$ inches broad, and will project 4 inches above the general surface of the body.

Palpation.—It feels quite hard and firm; no pain on pressure. The tumour can be felt not to be made up of one large mass, but several. There is a margin towards the thigh, and on deep palpation, pushing hand well into abdomen, the tumour edge can be felt to turn. The upper and outer part is doubtful; an indistinct margin can be felt, evidently beneath some of the muscle layers; but a hardness seems to exist further out, and whether this be a psoas muscle or not (for on flexing and extending the thigh a change is felt) cannot be distinctly made out. The lower margin

passes into the scrotum, and is much narrower. Only one-third of the scrotal mass is of the same consistence as the rest of the tumour; the remainder is much softer in consistence. The testicle, though distinct from this, feeling harder than normal, and cannot be altogether separated from it. Between these two, which are distinct from one another, though of the same tumour, several nodules of cartilaginous consistence can be felt. On percussing over the tumour the note is not flat all over, but along its whole abdominal margin a portion, about 2½ inches broad, gives a deeply intestinal note.

Operation.—On 10th September a long incision was made over the tumour above the reddish nodules. Inflammatory adhesions were found binding down skin to the tumour. The tendon of the external oblique muscle covered the tumour. This was divided, and the tumour capsule exposed. The upper part of the tumour was now examined to see how far the tumour extended, and if it was possible to remove it. A distinct rounded encapsuled margin was felt. The abdominal side of the tumour was now examined, and found not so encapsuled, and thick adhesions passed from the tumour to the tendinous structure. Another sweeping incision was made, joining the extremities of the former, and enclosing an elliptical area of skin in which were the reddish nodules. The skin was dissected down to the lower border of the tumour. The upper border was again attacked, and thick inflammatory adhesions cut through. Part of the conjoined tendon cut. Now entered the sheath of the rectus muscle; as now only transversalis fascia between the peritoneum and tumour, was thought best now to attack the scrotal part. An incision was made from the long incision over the scrotum. The whole half of the scrotum, tumour, with testicle was rapidly and easily removed from the scrotum. Thus hoped easier to reach the lower surface by raising the whole tumour. It now seemed as if the tumour followed the cord and passed into the abdominal cavity. Again went to the upper extremity. On dissecting, a further rounded, well-encapsuled tumour deeper and higher than the former was found at the upper extremity. It was about 4 inches broad. On raising and dissecting this, found peritoneum attached and adherent. It was dissected off as far as possible, then it was necessary to cut it, and enter peritoneal cavity. The intestines and omentum were now apparent, and kept back with a sponge while the tumour was further attacked. There now remained the huge mass attached to the deeper parts by pedicle. It was thought easier to finish by cutting through this, and thus be able to deal with deeper. The tumour being non-vascular in character, this was done. With the finger in the peritoneal cavity the tumour was felt to extend into the abdominal cavity and pass along the pelvic brim. The deeper part was gradually freed from its attachments. Several large veins were seen; one especially large seemed not to be femoral, though just

passing into it. After further dissection, the tumour was narrowed to a neck or pedicle about 2 inches broad; this connected the tumour with the deep parts. This was swept across, and the intra-pelvic part left alone. Carefully washed peritoneum and bowel exposed; stitched the edges of the peritoneum carefully together; then well washed the parts with corrosive; drainage—stitched—dressed.

Microscopical Examination.—Sections cut from this tumour were not of uniform structure throughout—the greater portion had the structure of wavy fibrous tissue with a large proportion of extremely flattened nucleated cells. There were also areas where the intercellular substance was much less fibrillated, being almost granular; in these areas the cells were extremely long and approached the type of those of a spindle-celled sarcoma. The bloodvessels, especially in certain small areas where the intercellular substance was almost absent, had no properly formed wall. Judging from by far the greater portion of this tumour, there need be no hesitation in pronouncing it to be a fibroma; but on examination of the above-mentioned small areas, one is left in doubt whether it is or not a spindle-celled sarcoma.

Progress.—For four days his temperature remained above normal. He complained of feverishness and flatulence. His bladder had to be relieved by a catheter once. On the fifth day his temperature fell, and since has never been above normal. The dressings were renewed daily, so that there might be no danger of urine finding its way to the wound. The skin did not completely cover on the gap left. In a few days healthy granulations covered the area. Healing satisfactory.

IV. Original Communications.

1. THE SHOULDER-JOINT IN RELATION TO CERTAIN DISLOCATIONS AND FRACTURES.

By Francis M. Caird, M.B., F.R.C.S. Ed., Lecturer on Surgery, Edinburgh School of Medicine (Report from the Surgical Laboratory, University of Edinburgh).

In our surgical text-books and monographs attention is given to the alteration in shape which the articulation of the shoulder-joint undergoes in old cases of dislocation. Such changes have usually been ascribed to absorption caused by disturbed pressure relations consequent on the new position of the bony surfaces concerned. Alterations and irregularities seen in the glenoid fossa of the scapula have been figured and described as occurring after such injuries, but the similar conditions of the head of the humerus are not so frequently referred to.

In his well-known paper (*Trans. Path. Soc. London*, vol. xii.,

1861) Mr Flower recognises Malgaigne's two forms of dislocation forward, but states that numerous intermediate degrees are to be found, and he summarizes as follows:—"The changes which take place in the contiguous extremities of the two bones in the ordinary forms of dislocation when left long unreduced, can be well studied in these specimens, and deserve more attention than has hitherto been paid to them, as they have frequently been mistaken for evidences of the existence of partial or pathological luxations. A new shallow socket is found upon the anterior surface of the neck of the scapula partly by absorption of old and partly by deposit of new bone around its edge. The exact position of this socket varies according to the degree of displacement of the humerus. In the first variety (subcoracoid of Malgaigne) the new cavity is formed more or less at the expense of the anterior portion of the glenoid fossa, which is gradually worn away, so that in some cases the original socket is almost altogether lost. A corresponding change takes place in the head of the humerus. Where it rests upon the edge of the glenoid fossa absorption occurs, so that a groove is excavated usually between the articular head and the great tuberosity. With continued friction this groove goes on increasing in size simultaneously with the changes in the scapula, the two accommodate themselves to each other, and ultimately the head of the latter bone presents a double articular surface, separated by a vertical ridge; the posterior part being part of the old glenoid cavity, the anterior the newly-formed socket. These respectively articulate with the sides of a wide groove placed vertically on the head of the humerus, and a rude kind of joint, allowing of a certain amount of motion, is the result. At the same time, it will be observed that by the absorption that has taken place in both bones the head of the humerus has made considerable progress towards regaining the position it occupied before the injury, and therefore the external signs of dislocation become to some extent removed. The under surface of the coracoid process, especially near its tip, is almost always found smooth and eburnated, having entered into the formation of the new articulation. If the head of the humerus is placed further under the coracoid process (intra-coracoid of Malgaigne), the new socket is formed upon the neck of scapula quite outside the glenoid fossa, upon which it does not encroach (Pl. II., fig. 4). It is then a simple cup-shaped depression, and the head of the humerus undergoes none of the changes above described, but is generally worn away on the side of the greater tuberosity by friction against the anterior edge of the glenoid fossa, and hence assumes a somewhat oval shape. In these cases the coracoid is not usually a part of the new articulation. All intermediate varieties are found between the two extremes. The position of the new socket, quite free from, or more or less encroaching upon the glenoid fossa, the head of the humerus unaltered, flattened on

its outer side, or grooved, being circumstances which all depend upon the exact situation in which the bone has found its new lodgment. The osseous surfaces, which were in contact, are in long-standing cases generally divested of cartilage, and in places hard and polished." There is no mention here of any initial injury sustained by the bones, and the glenoid fossa receives the chief attention.

Mr Flower presents us with a series of outline diagrams (Plate II.) of the glenoid and head of the humerus in section, to show the relative position of each when normal or with chronic rheumatic arthritis, or in the recent and old-standing dislocations. Going over his paper as far as the condition of the preparations admitted of exact description, we may easily recognise Malgaigne's dual types; the subcoracoid, distinguished broadly as follows: head of humerus grooved lying under and touching the coracoid process, glenoid encroached upon by the new socket. Intra-coracoid head of humerus unchanged, lying below and rather internal to the coracoid, which it only touches at the root, if at all. Glenoid unaltered, new socket distinct. Clinically the position of the head of the humerus is important, as also the increased tilting of the arm from the side said to be seen in the former injury. It may be observed, again, that in Mr Flower's paper the changes are ascribed to absorption from changed pressure relations. So also in Holmes' *System of Surgery*, third edition. That this is true as regards the intermediate types there can be little doubt. The element of time must also be considered. But the chief factor is the condition of the displaced head and its relations.

If we look at the outline diagrams (Pl. II.), we are tempted to ask how it is that in a recent subcoracoid dislocation the displaced humerus is able to remain balanced on the anterior lip of the glenoid fossa, even allowing that the slight depression at the anatomical neck does become engaged there. And further, if we seek an actual specimen illustrative of such a condition amongst the thirty-three preparations brought forward by Mr Flower, we find but one recent example, which, however, does not help us. In fact, such a state of affairs would almost indicate a partial dislocation, and could hardly be maintained for any length of time. In Ashhurst's *Encyclopædia* we read, "It is said that, in spite of all assertions to the contrary, that a true partial dislocation forwards occurs in consequence of the bone slipping forwards to the coracoid process, and the biceps tendon gliding back behind the head of the humerus, and by its tension holding it forward against the coracoid, although the anatomy of the part is such that the head could never rest in this position except for the support of the displaced tendon behind."

An examination of recent cases of subcoracoid dislocation casts much light on the whole subject. We are thus enabled to understand the grooving of the head of the humerus, the causation of

fractures above the level of its surgical neck, and also how injury to the glenoid and neck of the scapula may arise. Recent examples are not readily obtained. There are two in the collection of the University of Edinburgh, unfortunately without history or notes, which Professor Sir William Turner has kindly given for examination (Pls. I. and II.)

No. 168 is a dissected preparation in spirit of a recent subcoracoid dislocation of the shoulder, with indented fracture of the head of the humerus, produced by impact against the anterior lip of the glenoid fossa. The glenoid is rather bruised, the ligament along the anterior lip, especially at its lower part, is lacerated and pushed backwards. The capsule has apparently been ruptured at its lower and anterior part, and is pushed upwards. There is also evidence of rupture at the attachment to the lower portion of the glenoid fossa. The head of the humerus is rotated outwards, and is carried upwards, inwards, and forwards, so that the coracoid impinges against its upper surface. It shows a deep recent fracture indentation, which stretches from one point of the anatomical neck to another, mapping out a small segment of the cartilaginous head of the humerus on its posterior internal aspect. Into this groove, which measures about $1\frac{1}{2}$ inch in length, $\frac{1}{2}$ inch in breadth, and $\frac{1}{4}$ inch in depth, the anterior lip of the glenoid accurately fits. The posterior and lower part of the great tuberosity occupies the lower part of the glenoid fossa. The inner border of the lesser tuberosity is in line with the tip of the coracoid produced onwards. The bicipital groove, owing to the rotation, looks outwards and backwards. The tendon of the biceps was intact. The other muscles have been cleared away (see Plate I.)

167. Dried specimen of unreduced subcoracoid dislocation of the shoulder, with indented fracture of the head of the humerus, from impaction against the anterior lip of the glenoid fossa. This consists of the entire scapula, upper third of humerus, and outer third of clavicle. A small portion of the inner lip of the glenoid has been chipped off and bruised. The glenoid cartilage is pushed backwards. The capsule is represented by its anterior and superior parts attached to the scapula and humerus. A few bony nodules about the size of lentils are to be found adhering to the capsule at the base of the great tuberosity. The head of the humerus presents a large indentation, which evidently corresponds to the anterior edge of the glenoid. It is about one inch long, half an inch in depth, and is crescentic in form, with its concavity towards the root of the great tuberosity. When laid in position we note that the head of the humerus is thrown forwards, inwards, and downwards, and is also so rotated outwards that the bicipital groove corresponds to the axis of the coracoid process produced onwards (Plate II.)

There is a cast in the museum of this case prior to dissection (Plate III.)

The next case is extracted from the *Catalogue of St Bartholomew's Hospital Museum*, vol. i. p. 146.

No. 1019.—Dissection of a recently dissected shoulder-joint, which was reduced during life. The deltoid is cut across and reflected; the lower part of the muscle was bruised, and separated from the bone to a slight extent. The subscapularis is cut across about an inch from its insertion; its under surface at this point was slightly bruised. Neither this muscle nor the supraspinatus or infraspinatus was lacerated. The capsule of the joint was untorn; a small extent of its anterior attachment with the periosteum, with which it was continuous, was detached from the margin of the glenoid cavity and adjacent bone, *but the joint was not opened.* The capsule has been cut across at its anterior attachment. There is a deep vertical indentation or groove at the posterior margin of the articular surface of the head of the humerus, into which the anterior margin of the glenoid cavity accurately fits. It appears to have been produced by the violent impact of the head against the prominent rim on which it probably lodged.

From a man, æt. 36, who was knocked down by a train whilst at work on the line. On admittance he was found to have a subcoracoid dislocation of the right shoulder, in addition to numerous other injuries, from which he died about twelve hours after the accident. The head of the right humerus could be distinctly felt beneath the coracoid process. The axis of the bone was directed considerably outwards and backwards. Reduction was effected extremely easily, on slight traction being made in the usual manner. See paper by Mr Eve, *Med. Chir. Trans.*, vol. lxiii., 1886, p. 317.

We are in this way led to believe that the distortion dates from the moment of the receipt of injury. It would seem that the initial grooving takes place in the head of the humerus, which is indented by impact with the sharp anterior edge of the glenoid. The humerus becomes rotated outwards, and its head thrown forwards, so that the anatomical neck lands upon the anterior lip of the glenoid, which in its turn cuts deeply into the head of the humerus, as the patient falls with great force or receives a severe blow on his shoulder. This may occur with or without laceration of the capsule, the head of the bone passing directly forwards. Or, again, the head may clear the edge of the glenoid, or, rather, undergoing momentary displacement into the axilla, may then rise and occupy the intracoracoid position. Or, yet again, the impact between glenoid and humerus may be so great that one or other suffers fracture with or without the capsule giving way. An atrophied condition of the posterior portion of the head of the humerus is sometimes met with. This also might predispose to fracture or dislocation without rupture of the capsule, the head being more readily driven over the glenoid margin.

We can produce, although with some difficulty, and in a very artificial manner, a similar injury on the cadaver. Make a sub-

coracoid dislocation, lay the subject prone, and strike the scapula violently. It will be found that a series of lesions may be obtained, varying in degree from a mere bruise of the glenoid cartilage onwards to indentation of the head of the humerus or to complete intracapsular fracture, the anterior lip of the glenoid, hard and dense, cutting into the cancellated tissue of the humerus like a knife.

In evidence of this being the correct view, we may refer to some papers and observations which seem to have escaped the notice of general writers on surgery, and whose accuracy may thus be corroborated.

Malgaigne himself asks (*Traité des Fractures*, p. 497) if the grooves in the humerus are not frequently produced just at the moment of dislocation by the crushing of the head of the humerus against the border of the glenoid. He looked on such cases rather as examples of incomplete luxation.

Mr Joseph Bell, in his paper on the "Nomenclature of Scapulo-Humeral Dislocations" (*Ed. Med. Journal*, May 1863), quotes a case in which "the diagnosis was made of dislocation forwards, complicated with a longitudinal fracture separating the greater tuberosity from the head and the lesser tuberosity. It was one of those cases where, in addition to the primary dislocation, there is a consecutive fissure of the head of the humerus. How is this fissure produced? Not by any peculiarity in the direct violence which caused the injury, not by any conceivable action of the muscles, but by the sharp edge of the glenoid border being forced as a wedge against the posterior groove of the humerus. This is I hold to be the only rational manner in which we can explain such a complicated lesion. The position of the groove" (he alludes to the posterior sulcus of the anatomical neck) "of the humerus on the groove in dislocation explains in a most satisfactory manner the causation of those not uncommon but hitherto rather inexplicable injuries, I mean fractures of the anatomical neck of the humerus."

At the Cambridge meeting of the British Medical Association in 1880 (*Brit. Med. Jour.*, 1880, p. 349), Professor Bennet, of Dublin, brought forward five examples of dislocation at the shoulder complicated by fracture of the upper extremity of the humerus. One was recent, and the fracture incomplete, the others were united. From a study of these and other recorded cases, Professor Bennet states,—1. Fracture of the upper extremity of the humerus occurring as a complication of the dislocation commences at that part of the anatomical neck which rests, after dislocation has taken place, against the upper border of the glenoid cavity. 2. It is caused by pressure of the humerus against the sharp edge of the glenoid cavity, probably the result of a constrained position, preventing the separation of the elbow from the side as in ordinary dislocations. 3. While the fracture starts at the anatomical neck,

and may follow it strictly, it commonly passes into the shaft detaching the lesser tuberosity with the head. 4. The fracture is neither comminuted nor impacted.

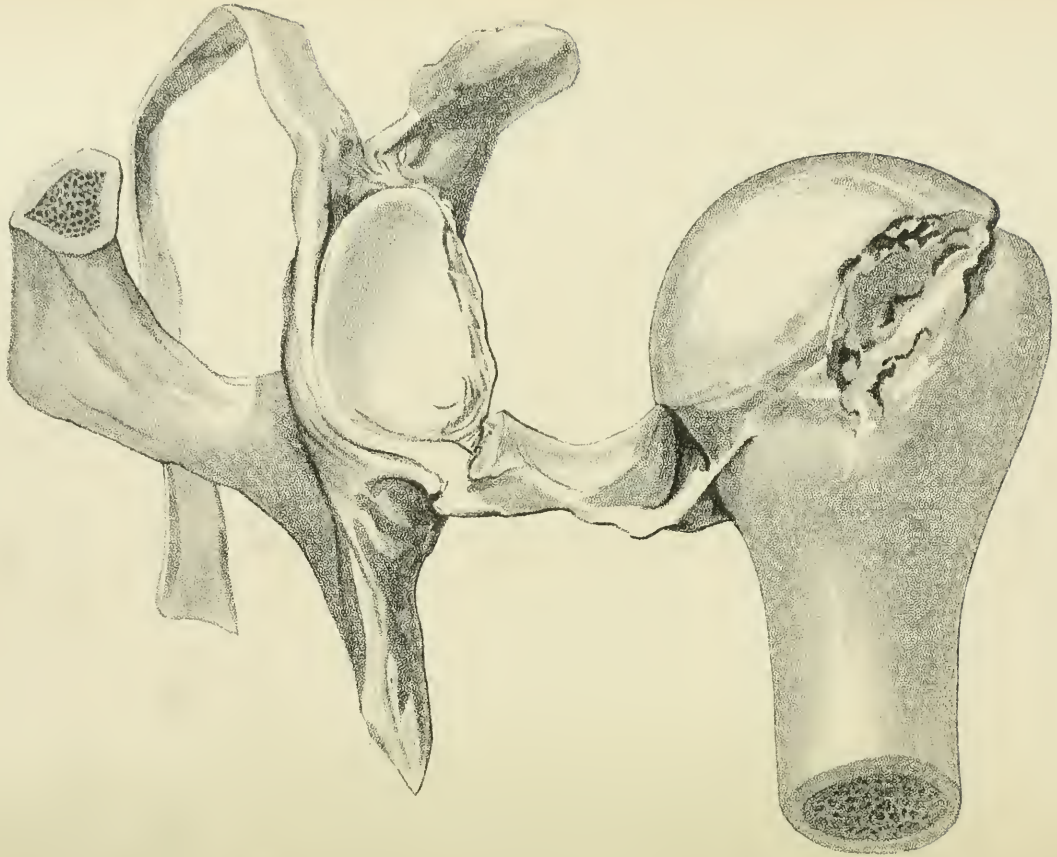
We have already made reference to Mr Eve's case, and now add a few sentences from his most suggestive paper. In the description he points out that the joint had not been opened into, although the capsule was stripped from off the anterior border of the glenoid cavity, remaining, however, continuous with the periosteum. He says,—"I have been unable to find on record a single case of traumatic dislocation of the humerus in which the joint was not opened. . . . . The indentation or groove on the posterior surface of the head of the humerus is another unusual feature in the case. The formation of the groove partially accounts for the slight damage to the capsule, as the head of the bone, having lodged on the margin of the glenoid cavity, was thus prevented from passing further inwards on the surface of the scapula. The groove was situated at the extreme margin of the articular surface, showing that the head was entirely separated from the glenoid cavity. The case cannot, therefore, be regarded as an instance of incomplete dislocation. . . . . The occurrence of such a groove as that observed in the case is of considerable interest, since it may explain the mode of production of fracture of the anatomical neck, with dislocation of the head of the humerus forwards. Further, the mode of production of the hitherto unaccountable cases of impacted fracture of the anatomical neck may perhaps, *in some instances*, be explained in a similar manner."

It would appear, therefore, that we are justified in recognising the true type of subcoracoid dislocation as being *always* associated with an indentation fracture of the head of the humerus caused by the dense, hard, anterior lip of the glenoid. The capsule may be ruptured or not. If there be complete fracture, the capsule may remain intact, retaining the upper fragment, or the capsule may give way, and the displaced fragment remain outside.

The subcoracoid dislocation lends itself specially to Koch's manipulative procedure for dislocations. Old standing cases are, however, not to be regarded with favour, for even should we get the head freed from adhesions, there is no longer any glenoid to receive it, atrophy taking place rapidly. With the *intra-coracoid*, however, we have greater hope of success, since here the glenoid remains comparatively unaltered for long.

From the fact that our museums contain many examples of dislocation forwards, we are apt to imagine that the downward displacement is much rarer. One must remember, however, that this may mean no more than a relative difficulty in reduction. Of nine *recent* cases admitted lately to the Royal Infirmary, only one was subcoracoid, eight were clearly subglenoid.

A careful description of the cause of the injury, the condition of the muscles and joints on the normal and abnormal sides, the



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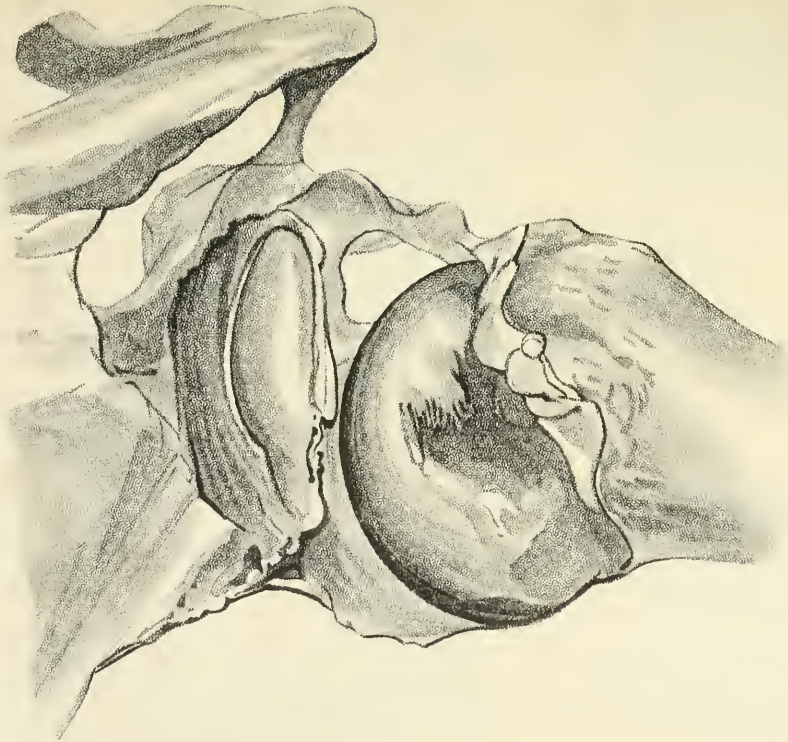

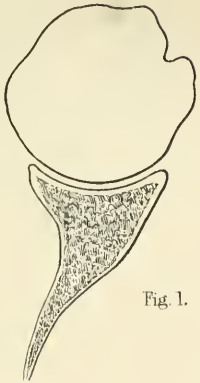

Fig. 1.

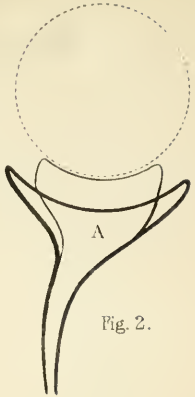

Fig. 2.

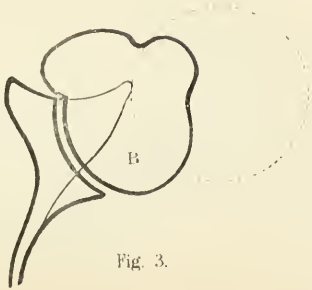

Fig. 3.

Fig. 4.

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PLATE III.

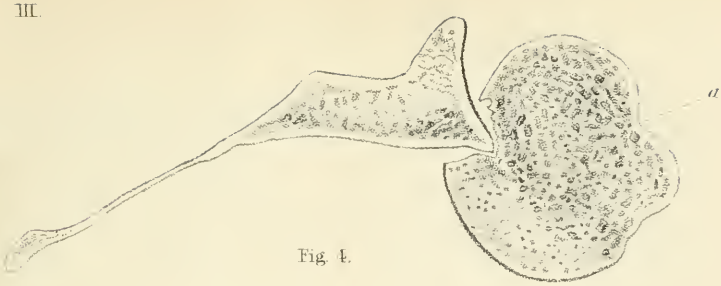

Fig. 4.

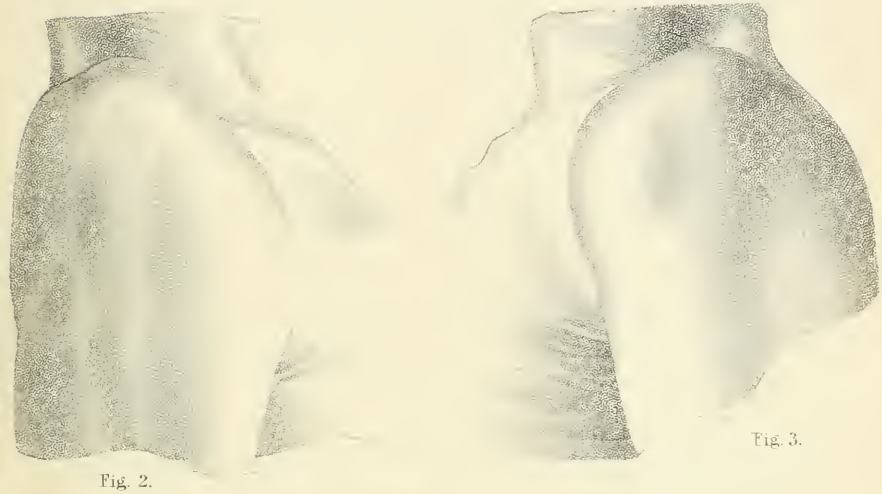

Fig. 3.

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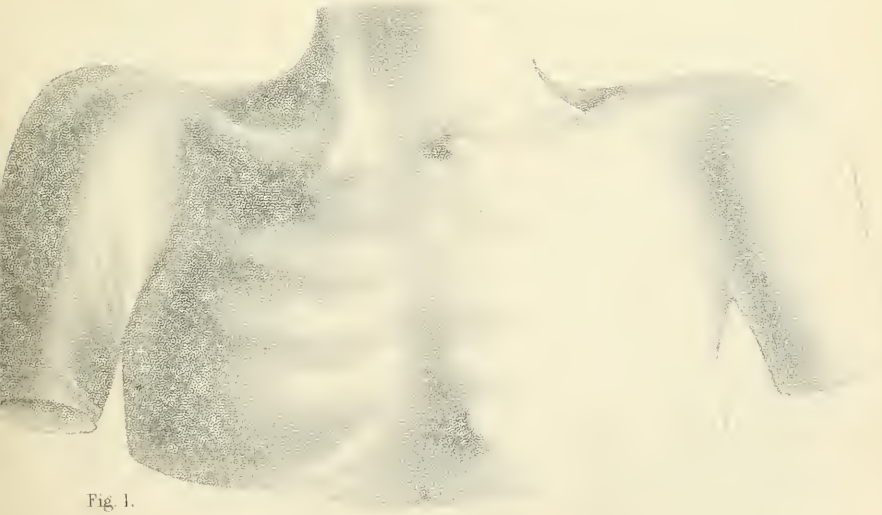


Fig. 1.

appearance of the shoulder as regards outline and projection, the amount of tilting and rotation, the precise position of the head of the humerus and its relation to the coracoid process, the presence of crepitus, and the history as regards rheumatic arthritis before and after injury, are all points worthy of careful investigation and record. We should not only be able to make an exact diagnosis, but at once to appreciate the form of manipulative treatment best adapted for each case.

Recent examples of fractures of the head of the humerus, as described from old-standing cases by W. M. Smith; fractures of the glenoid; longitudinal splits in the line of the biceps tendon, as diagnosed by Ogston and others, are still desiderata.

Description of Plates.

Plate I.—Recent subcoracoid dislocation of the shoulder-joint, with indented fracture of the head of the humerus. No. 168, Museum, Univ., Edin.

Plate II.—Subcoracoid dislocation, with indented fracture of the head of the humerus. No. 167, Mus., Univ., Edin. For appearance prior to dissection see Plate III.

Outline Diagrams from Mr Flower's Paper :—

Fig. 1.—Normal conditions of head of humerus and glenoid of scapula.

Fig. 2.—A, Glenoid as altered by chronic rheumatic arthritis.

Fig. 3.—Position of the head in recent subcoracoid dislocations. B, The same when left long unreduced.

Fig. 4.—Position of the head and formation of new socket in intracoracoid dislocation.

Plate III.—Figs. 1, 2, 3.—Anterior and lateral aspects of shoulder, as seen in case (No. 167, Plate II.), of subcoracoid dislocation prior to dissection.

Fig. 4.—Relation of glenoid and head of humerus, as seen in section, after artificial production of subcoracoid dislocation, with indented fracture of head of humerus.

Mr Joseph Bell, in the course of a few remarks, said that Mr Caird's paper was a very interesting one, and was the outcome of a very large amount of really good work on the subject. To himself it was a source of much pleasure to find that Mr Caird had not only read with care a paper on the nomenclature of scapulo-humeral dislocations written by himself twenty years ago, but that he also agreed with many of the conclusions of that paper. One of the chief points of interest Mr Caird had emphasized was the question of the dent in the head of the humerus. He did not think surgeons had been sufficiently alive to this as stopping short of fracture. He had held and taught that the impact on the edge of the glenoid was the cause of fracture of the anatomical neck.

Professor Chiene said that, though Mr Bell and Mr Eves had forestalled Mr Caird in regard to the causation of fracture by impact against the edge of the glenoid, he had shown that undoubtedly dislocation of the shoulder-joint might occur directly forwards. The old view that it was always first downwards and

then forwards he was now convinced was incorrect as regards subcoracoid dislocations. It was also evident that in old-standing subcoracoid dislocations surgeons were not justified in interfering to the same extent as in intra-coracoid dislocations.

Mr Caird said that, in looking over German literature, he had not hitherto found attention drawn to this point. He had to thank the University authorities, particularly Professor Chiene and Sir William Turner, for assistance and access to museum specimens; and also the Curator of the Museum of the Royal College of Surgeons.

2. ON A CASE OF POISONING BY CORROSIVE SUBLIMATE.

By Henry M. Church, M.D., B.Sc., F.R.C.P. Ed.

In all works on Medical Jurisprudence, cases of poisoning by corrosive sublimate are recorded. The cases, however, are not numerous. According to Taylor the smallest dose that resulted in death was two grains, taken by a child; and the largest quantity taken by an adult, that did not cause death, was 80 grains. Christison in his work *On Poisons* records a still wider range of susceptibility on the one hand, and on the other, resistance to the action of the poison. According to him, salivation and even death may occur from the smallest doses of mercury. Every general practitioner must acknowledge the numerous idiosyncrasies that meet him in the prescribing of any mercurial preparation. Hence it is reasonable to suppose that the poisonous dose must in like manner vary considerably. In our own city Dr Littlejohn informs me that within the last thirty years he had met with only two cases of corrosive sublimate poisoning (the fatal dose in neither have I ascertained) until this summer, when two fresh cases have been reported, one of which is the case I am going to relate. It has, as will be seen, a special medico-legal interest, on account of absence of mercury in the system after death.

In the following case of accidental poisoning a glycerine solution of corrosive sublimate was given for a dose of castor oil. The exhibited phial contains the substance. Though the colour is whitish, the physical properties are not unlike those of castor oil. After the usual training at the Maternity Hospital, a nurse, the mother of the unfortunate child, got with her, in a bottle which she did not label, a little of this substance. It was used at the Hospital as an aseptic for the nurses' hands, a little being largely diluted with water for washing purposes. The concentrated solution was prepared by Messrs J. F. Macfarlan & Co., and consisted of 86 grains of perchloride of mercury in one ounce of glycerine. As nearly as it is possible to estimate, the little girl swallowed

one drachm of this concentrated preparation—that is to say, about $10\frac{3}{4}$ grains of corrosive sublimate.

On May 7th, about 4 P.M., A. B., aged 5 years and 3 months, had administered to her a dessert spoonful of this poisonous substance. The spoon was put well to the back of the tongue, and emptied. The child, discovering the error, rejected at once what she could of the exceedingly acrid substance, but seems to have swallowed about one-half. From the mother's statements it appears that violent vomiting came on immediately, and that the stomach was largely emptied of its contents; this was followed by constant retching of mucus, which soon became tinged with blood. The bowels had also acted once or twice.

Four hours after swallowing the poison, when first seen by me, she had a pale ashy-coloured face, with very anxious expression. She had swelling of the lips and cheek, complained of pain in the throat and in the pit of the stomach, to which she frequently put her hand, and continued retching small quantities of mucus brightly tinged with blood. She asked constantly for water, and swallowed freely milk and white of egg. Pulse 108, fairly strong. Till 1 A.M. she remained much in the same state, and then fell into a drowsy condition till 5 A.M., when retching and purging came on. About 6 A.M. she got up, walked across the floor, put her mouth to the water tap, and drank freely during the temporary absence of her mother from the room. She fell again into a drowsy state for about an hour and a half, and then the previous symptoms returned with great virulence. At 10 A.M. her pulse was 180, very weak; respirations, 64; temperature in axilla, 105°·6, and in the rectum, 106°·6. During the night she had taken milk and the albumen of eight eggs. She seemed free from pain, but had terrors, such as a stone wall about to fall upon her, people coming to take her away, etc., also incoherent muttering about her playthings. The pupils were widely dilated and insensible to light. The most striking feature was a constant twitching of the eyelids and eyeballs, the latter being rotated upwards and kept upwards in a jerky manner. The fingers scratched rather than picked the bed-clothes, and every few minutes twitchings and tremblings of the whole body occurred, and at longer intervals violent startings. There was no salivation, but profuse mucous discharge from the nose. A little bloody mucus was occasionally discharged from the bowel. There was complete suppression of urine. Her mother states that she continued much in the same condition, with the eyes jerkingly fixed on the corner of the ceiling, till about 2 P.M., when she said, "I am dying," and in three minutes expired, exactly twenty-two hours after taking the poison.

Dr Littlejohn very kindly performed the post-mortem examination twenty-three hours after death, with the following results:—Lips livid; gums white; sordes on upper incisors; post-mortem rigidity and lividity well marked; features placid; face slightly

congested. On moving head, a little grumous mucus escaped from the nostrils. Upon exposing the abdominal contents, the portion of stomach visible was of a bluish-gray appearance. The peritoneum was natural. Left lung was congested, with fully 1½ oz. of coloured serum in the pleural cavity. Right lung was also congested, with a smaller quantity of coloured fluid in its pleural cavity. In the pericardium there was about ʒss. of clear serum. The right auricle of the heart was distended with blood for the most part fluid; there was also a decolorized clot. The heart was perfectly healthy. The tongue was sodden, and the pharynx presented a general diphtheritic look. The gullet was distended. A ligature was applied to the gullet, about one inch from its upper extremity, and another to the pylorus—the stomach and gullet, with the tongue, larynx, and trachea attached, being removed to Dr Littlejohn's retiring room for careful examination. The gall-bladder was distended with normal bile. The liver was apparently healthy; spleen normal; kidneys congested; bladder empty.

In the duodenum there was no trace of congestion. The small intestines contained a good deal of flatus, presented no marked state of congestion, but there was a grayish-yellow appearance of the mucous membrane throughout. In the lower part of the bowel the mucous membrane was more of a greenish colour. The cæcum had fæculent contents, but was not congested. The transverse and descending colons were markedly contracted, but with the exception of a patch of congestion, about the size of a florin, in the sigmoid flexure, the mucous membrane of the large intestine was pale and apparently normal.

Upon examination of the *removed* parts, the stomach was found to be discoloured along its smaller curvature, the larger curvature being comparatively unstained. When opened along its lesser curvature, there stretched from the pylorus along the curvature a band, 3 inches in breadth, of intense congestion, of port-wine colour. This congestion stretched down in small patches towards the larger curvature. The rest of the stomach presented a yellowish-gray appearance. No abrasion of mucous membrane could be discovered, but along the larger curvature the mucous membrane appeared thin and diaphanous. The discoloration surrounded the entire cardiac end of the stomach, and abruptly terminated towards the œsophagus by a well-marked line of slight congestion. To within 1 inch of its upper extremity the œsophagus also presented a yellowish-gray appearance. Above this there was marked congestion, which became intense over each tonsil, on the epiglottis, and on either side of the rima glottidis. Gray patches of a diphtheritic nature covered the fauces. The trachea was markedly, and the larynx moderately, congested.

Seven specimens were removed from the body for chemical analysis, viz., the mucus from the nasal cavity, the contents of the

stomach, the contents of the duodenum, the serum removed from the pleural cavity, the ejecta from the bowels six hours before death, a portion of the liver, and one kidney.

These were taken to, and carefully examined by Mr Falconer King, the city analyst, and by Mr Hunter, with the remarkable result that not a trace of mercury could be detected. Reinsch's test was what was principally employed. This is used for the detection of arsenic as well as mercury. It consists in taking a little of the fluid suspected to contain the poison—in this case corrosive sublimate—or a suspected solid dissolved in distilled water, and adding about $\frac{1}{6}$ part of pure hydrochloric acid. Into this is put a thin plate of pure copper. If mercury is present, metallic mercury deposits on the copper, giving it a gray coating which cannot be rubbed off, but takes on a bright and reflecting surface by rubbing. Dr Falconer King kindly showed me the seven plates of copper corresponding to the seven analyses, and in none of them was there any change of colour on the surface of the copper. The suspected fluids were also volatilized by heat, nor could any trace of globules of mercury be seen. The iodide of potash and other delicate tests were employed, but with like negative results.

Though in cases of poisoning by corrosive sublimate mercury is generally found after death, there are other cases on record where it has not. Taylor, in his work on *Medical Jurisprudence*, says, "A person may die from the effects of corrosive sublimate, and no mercury may be found in the tissues. A case of this kind occurred some years since at Guy's Hospital; and another in which deceased died in fifteen days from a large dose of corrosive sublimate in whisky, has been reported by Geoghegan. On this occasion, although the local effects of the poison on the throat, stomach, and bowels were of an intense kind, the viscera, on careful analysis, yielded no trace of mercury; the metal had been entirely eliminated in fifteen days." And in his work *On Poisons* the same author says, "Corrosive sublimate is not always found in the stomach of persons poisoned by it, although from its readily combining with the mucous membrane it is more likely to be detected than arsenic. In a well-marked case which occurred to Mr Watson, where 2 drachms killed a person in six days, none was found on a chemical analysis of the contents. This may have been partly due to the length of time that may have elapsed. In a case in which 2 drachms were swallowed, and the man died in four days, no mercury was detected in the stomach or tissues."

I might quote other cases, but enough are given to suggest the difficulty of the question chemically, and at the same time point out the possible importance of the absence of the poison at the post-mortem examination from a medico-legal point of view. No explanation seems to have been given of this absence of the poison. Corrosive sublimate is an exceedingly soluble salt, and is also easily reduced in contact with the animal tissues. According to

its former property it might soon be dissolved and eliminated from the system; and, according to the second, might be reduced to metallic mercury, and by its weight escape from the bowel, or be imbedded in the form of small globules in the folds of the mucous membrane, and so evade detection. No doubt whatever change does take place after the poison is taken must depend much on the contents of the stomach, and also upon the antidotes administered. Christison in his work *On Poisons* writes, "It appears from the researches of M. Boullay, confirmed by those of Professor Orfila, that various vegetable fluids, extracts, fixed oils, volatile oils and resins, possess the power of decomposing corrosive sublimate. According to M. Boullay a part of the chlorine is gradually disengaged in the form of hydrochloric acid, and the salt is consequently converted into calomel, which is deposited in a state of mixture or combination with vegetable matter." At page 436 he adds, "Another important consideration is, that corrosive sublimate may be decomposed and reduced to the metallic state by the admixture of various substances either given at the same time or subsequently, and the longer the inspection is delayed, the more complete will be the decomposition which is accomplished. Iron, zinc, and other metals are the most active of these substances."

Among the older writers, when mercury was so much in vogue as a remedy, many curious instances of quicksilver being discharged from the body are given. There are instances of metallic mercury passing from the bowel, and others of its being found in the urine and sweat. Fourcroy relates the case of a gilder attacked with an eruption of little boils, in each of which was contained a globule of quicksilver. A case is also recorded where a patient had taken corrosive sublimate in doses of 1 grain a day for a month, and used a mercurial gargle, at the end of which time there was discovered a black sediment in the urine, which, when separated and dried, was found to consist of little globules of mercury.

In the case, the subject of this communication, the fatal issue no doubt depended both upon the primary corroding action of the poison upon the tissues, and upon systemic mercurial poisoning, the symptoms of which were so well marked. It would appear also that the portion of corrosive sublimate that had not been got rid of during the early period of vomiting, and which caused the poisoning, had either been dissolved and eliminated, or had been reduced to minute globules of mercury, which might escape observation, even with the aid of a pocket lens, at an ordinary post-mortem examination, however carefully conducted.

Dr Bramwell said Dr Church has so fully considered all parts of the case that he has left little or nothing to be said. The most important point seemed to be the remarkable fact that, although the child only survived twenty-two hours, no trace of the poison could be detected by careful chemical analysis. In the previous

cases of poisoning by corrosive sublimate in which no poison had been found in the tissues after death, the patient generally survived for some days, and it was reasonable to suppose that the poison might have been excreted from the body; but, in this case, it was much more difficult to accept the excretion theory.

Dr Craig observed that the quantities of irritant poisons which could be taken with or without producing fatal results could never be accurately determined, because no one could tell the amounts ejected by vomiting or got rid of through the bowels. The probability was that, in this case, the greater part was eliminated either upwards or downwards. They could not very well get rid of the difficulty on the theory that the perchloride of mercury was changed into another salt, because it could only be changed into another salt of mercury, and the tests were applied to find mercury.

Dr James asked if it was usual in cases of irritant poisoning proving rapidly fatal to have such a rise of temperature (106·6 F.) as that noticed by Dr Church in his case? This might be an important question in reference to diagnosis, as one would expect a fall at the commencement or during the collapse following a dose of such a substance.

Dr Church said that in the recorded cases there was no mention of an observation of the temperature.

Dr Bramwell suggested that the temperature was probably taken when the stage of collapse was over.

Meeting II.—December 1, 1886.

Professor GRAINGER STEWART, *President, in the Chair.*

I. ELECTION OF NEW MEMBER.

Alfred Bell Whitton, M.B., C.M., Banff, was elected an Ordinary Member of the Society.

II. EXHIBITION OF PATIENTS.

Dr Allan Jamieson showed—(1.) A case of VARICELLA GANGRÆNOSA, and in doing so remarked:—There are three peculiar and prominent symptoms in chicken-pox. These are—the tendency the eruption exhibits to renew itself over and over again in successive crops; the itchiness which accompanies the complaint; and the scars which remain more or less persistently as tokens of its having been passed through. The first of these is always shown pretty distinctly, and on it we depend in some measure in forming our diagnosis. The second has been demonstrated by Mr Hutchinson

to be not infrequently more than ephemeral, to be capable of enduring for a very considerable period of time, long after the originating cause has faded out of recognition. To this induced hyperæsthesia of the skin he has attached the name of varicella prurigo. A like exaggeration of the ulcerative propensity has been named by the same acute observer varicella gangrænosa. Cases of this latter development are rare, and the following one is not without interest:—A. M'L. was aged one year and nine months when he was brought to the Royal Infirmary on the 19th June 1886. His father had shortly before had his leg amputated below the knee for a sarcoma involving the ankle-joint. His mother was a healthy woman, who had been eleven times pregnant. Five children were born prematurely. One died of scarlet fever. Of the six which remain, one has had tubercular disease of the mesenteric glands, and one of the cervical glands has suppurated. Four others are in good health. There is no evidence whatever of any syphilitic taint. A. himself was apparently in good health when born, but began to pine when a month old, and since then has never been robust. He has signs of rickets in the state of the radius. Two months since he had measles, then hooping-cough, and when he had just begun to pick up a little, the rash of chicken-pox came out. None of the other children had varicella at the time, but it was pronounced chicken-pox by a doctor seen at one of the dispensaries. The eruption appeared about the 5th of June, and it had thus lasted a fortnight when I first saw it. It occupied the back, head, face, and right thigh. On the scalp and face were numerous small pustules. On the back in the centre were circular lesions elevated a line above the surface, rose-red at the edges, depressed in the centre and dark brown. There were also numerous ulcers, some as large as a shilling, with a red elevated margin, punched out edges, and a granular floor. Similar ones were also seen on the right thigh; all were circular. On the nates were flattish rose-red papules; some have had a vesicle at the summit, but this had dried into a crust. There were no clear watery vesicles when first seen. The eruption itched somewhat. Percussion resonance was impaired over the chest posteriorly, and there were some moist râles with prolonged expiration,—some degree, in fact, of broncho-pneumonia. Two days after this sketch of the back was made by Bayne. On 22nd June many fresh spots had come out on the back and face. The older ones had enlarged, and tended to ulcerate. The feet and hands, the lower part of the legs and arms, were still unaffected, and there were few spots on the abdomen. The child looked pale, and seemed to suffer much pain. It cried when touched or moved. Small doses of quinine and Dover's powder were prescribed. On the 24th some small clear vesicles with red areolæ had come out on the abdomen. More of those on the back had grown larger and ulcerated. Fresh crops of vesicles continued to appear over

the whole surface till the 16th July, many becoming transformed into ulcers. Diarrhœa set in, but was checked by milk and lime water, and the pretty free administration of port wine. By the 16th July there was a decided improvement in all respects. Cicatrization had taken place under the scabs which had formed on the ulcers, and on the head many of these had fallen off, and left smooth bald patches. *27th July.*—There were now merely many round depressed scars all over the body. The skin on these cicatrices was thin, pink, and translucent. Child now in fairly good condition. On the 19th of November many of the cicatrices had become smooth and very nearly level with the skin around. In nearly all the sound margin of skin continued pigmented, the trace yet unobliterated of the areola. Hair had grown on the scalp to a considerable extent. There persisted a good deal of itchiness of the surface. Mr Hutchinson[1] has aptly compared varicella gangrænosa with rupia. Both manifestations are to be regarded as proofs either of a severe form of the respective diseases, or of some idiosyncrasy. In a case recorded, with observations, by Dr Payne,[2] which ended fatally, acute miliary tubercles were found in the lung; and he quotes from Dr Barlow some facts which confirm the impression that this coincidence of tuberculosis and varicella gangrænosa is not entirely fortuitous. Though there was no direct evidence of tubercle in A. M'L's. case, unless the broncho-pneumonia partook of this diathetic character, there was proof of such a taint in the family. If any doubt existed on the subject in this country, this case serves to support the idea prevalent in Britain that varicella is not a mere modification of smallpox, as is taught to some extent in Germany. It is somewhat curious that a pruriginous rash and a gangrenous eruption has in rare cases followed vaccination,[3] but no link connecting these with tuberculosis has so far been furnished. (2.) A case of LUPUS EXFOLIATIVUS without ulceration in a young girl, which had lasted about fourteen years. It had been latterly treated with the salicylic and creasote plasters, the 10 to 20 and afterwards the 15 to 30 plasters having been applied. After the disease had been destroyed by them it was treated with zinc ichthyol salve muslin, and then with mercurial plaster. With the exception of one doubtful spot, which they were watching, the disease had quite disappeared.

III. Exhibition of Pathological Specimens.

1. *Dr Skene Keith* showed an OVARIAN TUMOUR of some interest, as the mass was situated outside the peritoneal cavity. The tumour, which weighed 17 lb., had grown from the left ovary, opening up the broad ligament, then passing across the pelvis

[1] *On Rare Diseases of the Skin*, p. 235 ; *Med. Chirurg. Trans.* 1882, p. 9.
[2] *Trans. of Path. Soc., Lond.*, 1885. [3] Hutchinson, *op. cit.*

behind the uterus, and splitting up the right broad ligament. After filling the pelvis, it had grown upwards, raising the uterus entirely into the abdomen, and the right ovary to the level of the umbilicus. It was thus necessary to divide the peritoneum twice before reaching the tumour. Mr Keith mentioned that this operation completed his first hundred cases of ovariotomy; and in answer to the President said that there had been three deaths and ninety-seven recoveries.

2. *Mr A. G. Miller* showed A LEG amputated for senile gangrene of the toes. The operation was performed through the lower third of the thigh because of the absence of pulsation in the tibial and popliteal arteries. Just where the femoral was cut through, a plug was found blocking up the vessel, and a little further up, where it was found to be pervious, a ligature was applied. Hutchinson advised amputation high up and early in such cases. The patient, a man, was doing well.

IV. Exhibition of Instrument, etc.

1. *Dr Foulis* showed a model of an ASPIRATING SYRINGE. It consisted of an ordinary syringe with two tubes fitted to the body of it. One of these communicated with the cavity, and when the piston was withdrawn served to suck air from a bottle which could be attached to the instrument. The other tube also communicated with the bottle, and by it the fluid was withdrawn which it was required to remove. The advantages claimed for it were the control it afforded over the atmospheric pressure, its simplicity, and cheapness.

2. *Dr Allan Jamieson* showed PILLS coated with keratin, which was insoluble in the acids of the gastric juice. Unna, when making investigations into the formation of keratin in the tissues, found that it could be made insoluble in the acids of the gastric juice. The shavings of horn were digested in artificial gastric juice, then treated with ammonia which was driven off, and a substance left behind soluble in spirit, and insoluble in hydrochloric or acids of the gastric juice. This was a most valuable property, for pills coated in this way were dissolved only after passing the stomach. The pill-mass must be covered with cocoa butter before being coated with the keratin. The pills shown were got from Hamburg, but could be made in this country. One of them, containing five minims of the oil of turpentine, was intended for cases of enteric fever in which there was flatulent distension. The other contained lead and opium, which Dr Jamieson proposed to use in certain cases of typhoid diarrhœa, in which the stools being alkaline he hoped for some benefit from the keratin coating.

V. Original Communications.

1. A CASE OF CEREBELLAR TUMOUR.

By George Leslie, M.B., F.R.S. Ed., Falkirk.

WITH PATHOLOGICAL REPORT.

By Byrom Bramwell, M.D., F.R.C.P. Ed.

On the 11th April 1885 I was called to attend Mr J. M., æt. 24, assistant to his father, who is a master in the building trade, residing at Falkirk. He complained of severe, almost constant, frontal and supra-orbital headache. I learned that up to the age of 20 he had enjoyed good health, but towards the end of 1880 he suffered from tonsillitis, and about the same time began to be subject to attacks of vomiting, and to feelings of torpor and general malaise. During 1881 vomiting was frequent, and by the early spring of 1882 it was very severe, resisting all remedial measures. In May of this year, however, he had sufficiently recovered to be able to go to sea, and he spent the most of the time, from May till October, cruising off the western coast and islands. He seemed to derive much benefit from this, the vomiting ceased, and he returned home apparently convalescent.

Soon after his return his mode of walking was observed to have changed; it was no longer perfectly steady and firm, but was somewhat lurching, suggestive of a sailor's land gait. No importance was attached to this, as it gave the patient no inconvenience, was not accompanied by giddiness, and it was supposed by his friends to be a habit acquired at sea. He was also observed to have a greater tendency to sleep, being often abnormally drowsy, and he could only with difficulty be roused in the morning.

In January 1884 he began to complain of frontal headache, which was aggravated by stooping or other movement, and by July it had become very severe. From this time, until the date of my first visit, headache had been almost continuous, was usually frontal, but was now sometimes also referred to the right occipital region, and it was the most prominent of his symptoms.

About the end of 1884 the peculiarity of gait became exaggerated, and his father informs me that he first observed a distinct staggering movement, as he walked behind him in January 1885. This staggering or reeling gait had been earlier noticed by his acquaintances, and had sometimes been attributed to causes which were not in operation.

His memory, which had never been very good, began to fail sensibly early in 1885, so that he frequently got into difficulties by forgetting business orders and instructions.

His social condition and surroundings had always been very favourable. There was no history of alcoholic or other form of intemperance, of syphilis, or of any serious personal accident or injury. His family history was good, and there was no suspicion of any inherited disease.

During my first examination I found that the external conformation and development of the patient was good. He was of average height, and his muscular system was very well developed. He was not confined to bed. The facial expression was dull, heavy, and pained, although his general aspect was that of intelligence. The temperature was normal.

In the nervous system the most obvious symptom was the cephalalgia, referred to the frontal region. The pain was described as being very severe, almost continuous during waking, and of a darting character. Vision was little impaired, but ophthalmoscopic examination revealed optic neuritis, more marked in the right eye, and in an early stage. The pupils were dilated, but were equal, and responded normally to light and to accommodation. The other sensory functions, the organic reflexes, the skin and tendon reflexes, exhibited no distinct abnormality. All his movements were performed slowly, and this was especially noticed in his measured and deliberate articulation.

While examining the motor functions, it was seen that locomotion was imperfect. He was able to walk fairly well, but his gait was unsteady, showing swerving or rolling side motions. No particular bias to either side could be detected; there was no appearance of rotatory movement. He was able to stand erect without discomfort with his eyes closed. A few days after my first examination I had an opportunity of seeing him walk on the street when he was unconscious of being medically observed. He was then walking slowly and carefully, but not quite steadily; he appeared to be exercising voluntary control over the muscles of locomotion. A casual observer might have noticed little or no peculiarity.

All my questions were answered by the patient with great clearness and intelligence; there was evidently absence of any mental affection. There was complaint of tendency to sleep in the early part of the day, of a feeling of torpor, and of impaired memory. Strict inquiry was made as to the occurrence of giddiness, but the patient assured me that this had never been experienced.

The tongue was coated with a yellowish fur; there was chronic hypertrophic tonsillitis. The appetite was stated to be large, sometimes excessive, and the bowels usually constipated. The urine was slightly phosphatic. In other respects physical examination did not reveal any noteworthy feature.

The diagnosis was that the symptoms were caused by a cerebellar tumour, and the prognosis was consequently unfavourable.

I prescribed 10 grs. potass. iodid., 15 grs. potass. bromid., with ½ oz. inf. chiratæ, to be taken three times a day. This was

continued for about a month, together with treatment for the constipation and tonsillitis. The effect was at first exceedingly beneficial—the headache, previously so continuous and severe, entirely ceased, and a feeling of wellbeing, which had been so long absent, was established. The patient was able to walk out daily, although still with the uncertain gait. About the beginning of May, however, the headache gradually returned, increasing in severity notwithstanding the continuance of treatment, and on the 14th May I brought him into Edinburgh for consultation. Dr Byrom Bramwell examined him, arrived at the same diagnosis, and asked me to continue the treatment directed to decrease the intracranial pressure.

For about a month after this, viz., till the 12th June, the symptoms were not very urgent, headache was not much complained of, and the gait had not visibly altered for the worse. On the 13th there was an attack of conjunctivitis which quickly disappeared under treatment, and about the 15th headache began to be very severe. On the 18th it was intense, and on this date vomiting, which had not occurred for three years, returned. It was frequent, was not accompanied with nausea, but came on without any warning. There was also an irritative cough which evidently did not depend on the state of the pulmonary viscera. During the short sleeps which were obtained respiration was seen to be much interfered with. The breathing was not typically cerebral, but the cycle usually contained a full, deep respiration, then a more shallow one, which was followed by a pause of from 30 to 50 seconds' duration. The treatment at this time was mainly palliative.

By the 22nd June the symptoms had so abated that the patient was able to get out of bed. It was then seen that the power of locomotion was much diminished, he could only walk while steadying himself by the wall or other support. The facial expression had somewhat changed, and it was, for some little time after this, vacant and staring. He was morose, and did not like to be noticed.

On the 24th June I was informed that there had been two general spasms during the preceding night, also clonic spasms of the left little finger, and strabismus. The headache was not now severe, and the vomiting and cough had ceased. On the 26th, at 3 A.M., during sleep, there were clonic spasms of the right arm for five minutes, and later in the morning complaint of cramp in the right leg. The breathing was normal, and headache was absent. On the 28th, at 4.30 A.M., there were clonic spasms of the right arm and leg. He was able to get out of bed, but walked with great difficulty. On the 29th giddiness was for the first time complained of, and was occasionally present for two or three days. In the beginning of July interference with the vaso-motor function was made apparent by an œdematous condition of the eyelids and the neighbouring tissues, and this continued, varying in degree, for about a

fortnight. On the 10th July headache, which had been absent for fourteen days, was again present, and also irritative coughing. On the 16th I was called at 6 A.M., and found the headache intense, and referred to the frontal and right occipital regions; there were violent tremors of the whole body, causing the bed to shake. Right tinnitus aurium was also complained of, and was compared to the sound of an engine blowing off steam. During this day cephalalgia was very severe, and vomiting very frequent. The patient only slept one hour.

By the 20th July the distressing symptoms had again almost disappeared, and the patient was soon able to get up every day. From this time until the middle of August frontal headache was felt for a few hours every morning, and there were occasional attacks of vomiting, but no spasms or interference with respiration. On the 17th August, and for a few days afterwards, he complained of a tendency to step or fall backwards when he was attempting to walk. On the 20th August I sent him to reside with friends in Avonbridge to try a change of air and scene, and after this I had less frequent opportunities of seeing him. The only new symptoms of importance which developed during the rest of his life—he died on 3rd November—were what were described as fainting attacks, varying in duration from one to five minutes, and which, as Dr Bramwell has suggested, were probably epileptoid.

Exacerbations of headache and vomiting took place at decreasing intervals, until within the last fortnight they occurred every two days. Optic neuritis had been latterly rapidly advancing and vision was very imperfect, but the patient could still see to shave until three weeks before his death. Hearing was always good. The mental functions never failed; only two days before his death he was able to remain downstairs for most of the day, and to converse intelligently. Death occurred half-an-hour after an attack of vomiting. There was euthanasia.

Dr Byrom Bramwell came to Falkirk for the post-mortem examination, and took the brain to Edinburgh. The following is his report on its pathology:—

The post-mortem examination was made on 6th November 1886; the head only was examined.

The scalp, skull cap, and dura were normal. The convolutions of the brain were flattened in an extreme degree and the sulci completely effaced; there was no evidence whatever of meningitis either at the base or the convexity—a point of interest in connexion with the extreme double optic neuritis which was present.

On slitting up the tentorium cerebelli, a cyst, fully the size of an ordinary orange, projected from the upper surface of the cerebellum, and seemed more especially to involve the middle and right lobes.

The wall of the cyst was in places extremely thin and fragile;

and the cavity of the cyst appeared—for it was not cut into—to be filled with a clear transparent limpid fluid.

The pons and medulla were flattened, compressed, and softened—the usual condition in large cerebellar (sub-tentorial) tumours.

The other parts of the brain appeared to be—and were on subsequent examination found to be—healthy. The ventricular system of the brain was dilated, though by no means to such an extreme degree as I have sometimes seen as the result of tumours of the middle lobe of the cerebellum.

The sub-vaginal spaces surrounding the optic nerves were distended with fluid; most marked optic neuritis could be seen with the naked eye in each fundus oculi.

With the object of having the brain photographed, and the appearance of the cerebellar cyst exactly reproduced, the specimen was not further examined at the time of the post-mortem, but was placed in a basin and taken by rail to Edinburgh. Unfortunately the jolting of the railway carriage proved too much for the thin wall of the cyst, for on arriving at home I found the cyst collapsed and its contents escaped.

On careful examination the middle lobe of the cerebellum was found to be almost entirely destroyed by a gliomatous tumour, in one part of which a flat, gritty, calcareous nodule, in diameter fully the size of a fourpenny-piece, was found. The tumour of the middle lobe of the cerebellum was situated in the posterior wall of the cyst, and there could be little doubt that the cyst was a secondary development of the tumour. The cyst had made its way under the superficial fibres of the upper surface of the right lobe of the cerebellum. The left lateral lobe was somewhat compressed but otherwise healthy; the right lateral lobe much compressed and somewhat atrophied; its upper surface being, as has been previously mentioned, invaded and destroyed, but only superficially, by the cyst.

On microscopical examination, the tumour was found to be a glio-sarcomatous tumour; in places the tumour tissue was remarkably vascular; numberless collections of hæmatoidin granules, the representatives of former blood extravasations, were scattered here and there throughout the tumour. In some parts of the tumour the walls of the vessel were enormously thickened and infiltrated with a clear, translucent, highly refractive material which stained pink with picro-carmine, and which was evidently the same hyaline material as that found in the walls of the gliomatous tumour of the left frontal lobe which was brought before the same meeting of the Medico-Chirurgical Society at which this paper was read, and which will be described in a future number of this Journal.

The optic papillæ were found, on microscopical examination, to be in an advanced stage of papillitis; the trunks of the optic nerves behind the lamina cribrosa also presented marked inflammatory

appearances, more especially in their peripheral portions, *i.e.*, just below the pial sheath. The sub-vaginal space was distended with what seemed to be inflammatory products (some red blood corpuscles, granular debris, and lymphoid cells); the connective tissue trabeculæ crossing the sub-vaginal space were much thickened

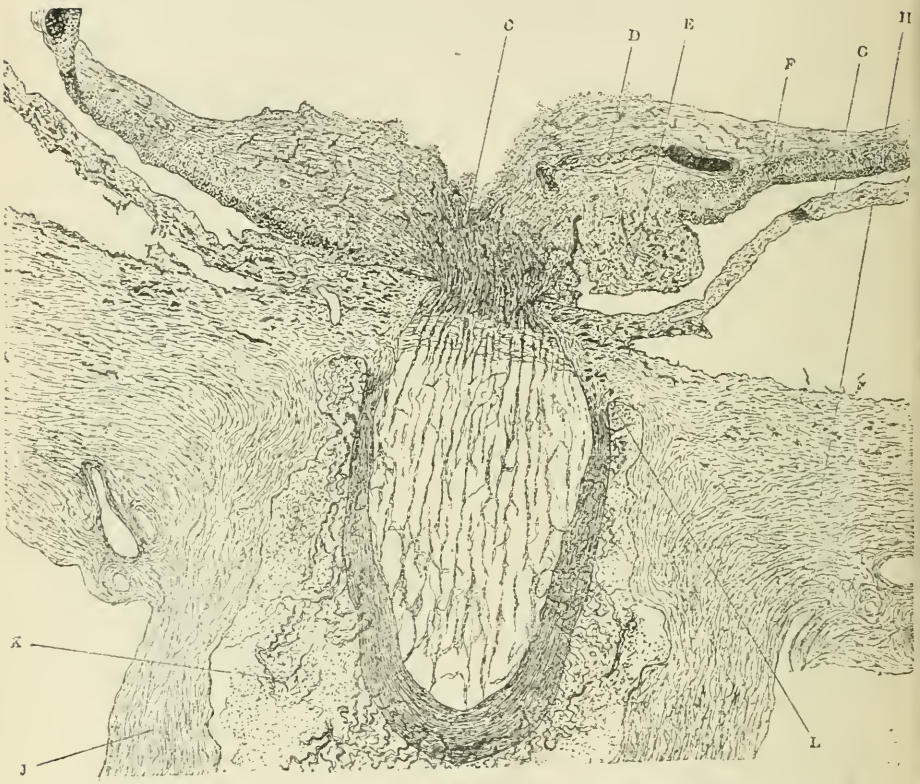


Fig. 1.—*Camera Lucida Drawing of a Section through the Optic Nerve and surrounding parts in Dr Leslie's case of Cerebellar Tumour, showing great swelling and vascularity of the optic papilla, the presence of inflammatory products in the sub-vaginal space, and swelling of the connective tissue trabeculæ surrounding the pial sheath. Stained with Picro-Carmine, cleared with absolute Alcohol and Oil of Cloves, and mounted in Zylol Balsam.*

C, centre of that portion of the optic papilla which is seen in this section; D, large vessel, which is in places distended with a dense mass of blood corpuscles; E, swelling of the deeper layers of the retina adjoining the optic papilla; F, deeper layers of the retina at a place where they are healthy; G, choroid, which has become detached from the retina and sclerotic; H, sclerotic coat; J, vaginal or dural sheath of the optic nerve; K, sub-vaginal space, situated between the dural and pial sheaths of the nerve, and which is distended with inflammatory products, right up to its termination (L).

and their nuclei very prominent. These appearances are well seen in the figure.

Remarks.—Among the points of interest which this case affords the following may be noticed:—(1.) The long duration of the

disease. There can be little doubt but that the cerebellar lesion preceded the vomiting attacks, which first appeared in the end of 1880, five years before death. Vomiting was an early as well as a late symptom, and, according to the description given by the patient, was of uniform character, and it latterly was of distinctly central origin; (2.) The mode of growth of the tumour, which could clinically be divided into three stages—the first, of onset, from autumn 1880 to summer 1882, corresponding to the period of tumour formation; the second, of comparative latency, to the beginning of 1884; and the third, of recurrence, from this time until death. During the third period there must have been rapid cyst formation, and consequently greatly increasing intra-cranial pressure; (3.) The severity of vomiting as a symptom of cerebellar disease, and which, together with the respiratory and vaso-motor symptoms, were doubtless the result of irritation of adjacent centres in the medulla; (4.) The almost complete absence of vertigo. In the treatment the administration of iodides, with a free use of laxatives, was often of great benefit—anti-syphilitics had no appreciable effect.

2. A CASE OF CEREBELLAR TUMOUR OF UNUSUAL CLINICAL AND PATHOLOGICAL INTEREST.

By Byrom Bramwell, M.D., F.R.C.P. (Edin.), Assistant Physician to the Edinburgh Royal Infirmary; Lecturer on the Principles and Practice of Medicine, and on Practical Medicine and Medical Diagnosis in the Extra-Academical School of Medicine, Edinburgh; Additional Examiner in Clinical Medicine in the University of Edinburgh, etc

On the 16th of October 1884 I was asked by Dr Foulis to see a single lady (Miss A.), aged 47, suffering from headache, vomiting, and partial loss of motor power in the right arm and leg.

At the age of 12 the patient had suffered from cerebral symptoms, the exact nature of which could not be definitely ascertained, but which were said to be due to "water on the brain." From this attack she had perfectly recovered, and had been an extremely intelligent, bright, and active-minded woman.

Ten years before the present symptoms commenced she had, after severe mental trouble and anxiety, another cerebral attack—a fit of some kind or another—attended with loss of consciousness, loss of speech, and marked rigidity of the limbs; the rigidity and speechlessness lasted for five days, during which time she was confined to bed. From this attack she had also (apparently) perfectly recovered, though her friends were conscious of a distinct difference in her mental condition, and had enjoyed good health until a little more than a year before I saw her.

At the age of 45, when menstruation ceased, she became anæmic, and began to suffer every now and again from attacks of headache

and vomiting. The headache was sometimes very severe, and always limited to the left side of the head; in the earlier periods of her illness it was apparently neuralgic in character, and very definitely located over the area of distribution of the great occipital nerve. For some time before I saw her the pain was chiefly felt in the left frontal and parietal regions.

The headache often occurred without the vomiting, but the vomiting was always associated with headache.

Under treatment the anæmia improved, the headache and vomiting became less frequent and less severe; but during the next few months she had several pseudo-apoplectic attacks which resembled a deep sleep, and in which she remained insensible for many (in one instance for twenty-four) hours at a time. One of these attacks occurred when she was in her bath. When seen by Dr Foulis shortly after the commencement of the coma she was completely insensible, the eyes suffused, and the temperature 105° F. During these attacks the contents of the bladder and rectum were evacuated involuntarily. The attacks of coma were never, so far as could be ascertained, preceded by a convulsion, and were not followed by any appreciable localized loss of motor power.

Some four months before I saw the patient, distinct loss of motor power had been occasionally noticed in the right arm and leg. The paralysis was never complete; it varied in degree from time to time, and would sometimes completely disappear.

A few days before my visit there had been distinct loss of power on the *left* side of the face (*i.e.*, on the opposite side to the paralysis of the arm and leg), and considerable difficulty in swallowing and articulating.

When seen by me on October 16th, the patient looked fairly well when lying in bed, though somewhat pale and anæmic. She could stand and walk, but with difficulty. After getting out of bed she looked very shaky and very ill, and the want of attention, vacant expression, and evident want of the power of mental concentration, which were very perceptible when she was lying in bed, became now much more marked. She seemed dazed, but said she was not giddy. Her movements were markedly unsteady, apparently the result of motor weakness rather than of inco-ordination. When standing she seemed very insecure, and it was noticed that the head tended to fall to the right side; this was particularly marked when she was seated in a chair having the eyes examined.

There was very decided loss of power in the right arm and leg; and voluntary movements of these (the paralyzed) parts were attended with a marked coarse tremor, which exactly resembled the tremor of multiple cerebro-spinal sclerosis, and which, it is well known, may result from pressure on motor conducting fibres.

The *left-sided* facial paralysis which had been noticed a few days

previously had disappeared; the motor power in both cheeks seemed feeble, but I was unable to satisfy myself that there was distinct localized paralysis on either side.

The tongue was protruded in a tremulous manner, and its tip, when protruded, seemed to be slightly turned to the right side.

There was marked difficulty in swallowing both solids and liquids, but there had been no regurgitation through the nose. Speech was distinctly impaired, apparently from slowing of articulation; there did not at this time, so far as could be ascertained, appear to be any definite aphasia.

The knee-jerk was lively on the left, but very feeble on the right (paralyzed) side; the plantar reflex was lively on both sides, but more marked (extremely active) on the left.

There was no apparent loss of sensibility; sight and hearing seemed perfect; the pupils were active, small, and contracted; ophthalmoscopic examination (which was made with difficulty at the first visit, owing to the small size of the pupils and the inability of the patient to keep the head steady and in the erect position, but which was most satisfactorily accomplished on November the 8th, when the patient was a great deal better) showed that there was no optic neuritis; both discs were deeply cupped, and perhaps rather too gray in colour, the edges well defined, the vessels of normal size; the sclerotic ring on the inner side of the left disc was very prominent, and a small white patch, very like one of the white spots seen in albuminuric retinitis, but in all probability the result of a previous hæmorrhage, was present on the inner side of the right disc. (Dr Barlow, who had seen the patient some months previously, had found some changes in the right disc, to which I will presently refer in detail.)

All the cerebral functions seemed slow and dull; but, so far as could be ascertained, there were not, at the time of my visit, any positive derangements of the mental and intellectual faculties. Both Dr Foulis and the patient's relatives had, however, on several previous occasions noticed marked evidence of intellectual failure (rambling and incoherence of speech, nonsensical talk, the erroneous use of words and expressions, impairment of memory, and marked loss of the power of attention).

Percussion did not seem to cause more pain over the left parietal and frontal regions than over the other portions of the cranium.

The urine had been repeatedly examined by Dr Foulis, and had always been healthy. There was no evidence of disease in any of the thoracic or abdominal viscera.

Diagnosis.—After careful consideration it was concluded—(1.) That the patient was suffering from an organic intra-cranial lesion; (2.) That, while the symptoms might be due either to a cerebral tumour or localized and progressive softening, the balance of evidence was very strongly in favour of tumour. A decided

opinion in favour of tumour was, therefore, expressed to the patient's friends.

The exact localization of the lesion presented very considerable difficulty. The right-sided character of the paralysis showed, without doubt, that the lesion was situated on the left side of the brain; while the presence of coarse tremor in the paralyzed limbs, and the intermittent character of the paralysis and tremor, were strongly suggestive of pressure on the motor (pyramidal) tract, rather than of a lesion directly involving and destroying the motor conducting fibres or the motor centres.

The fact that the *left* side of the face had been paralyzed was very strongly suggestive of a lesion involving the left side of the pons Varolii, and pressing upon the fibres of the pyramidal tract, going to the right arm and leg above its decussation in the medulla and on the trunk or fibres of the left facial nerve after their decussation in the pons. And that the lesion was situated far back in the neighbourhood of the pons or medulla seemed further confirmed by the marked difficulty in swallowing, by the character of the articulatory disturbances which were present at the time of my first visit. Further, the fact that in the earlier stages of the case the pain was limited to the back of the head, and most distinctly localized in the course of the great occipital nerve, seemed to favour this view of the position of the tumour.

The facts did not seem sufficient to warrant an opinion as to the pathological nature of the lesion.

Accordingly, when writing the same evening to Dr Barlow, who was interested in the case, I stated that I had no doubt that there was organic disease; that I was strongly in favour of a tumour rather than of softening; that the localization of the lesion was a matter of difficulty; but that, for the reasons which have been given above, I was inclined to think that it was situated far back, and was probably exerting pressure upon the pons Varolii. At the same time I stated that the mental defects, and I should have added (but I am not sure if I mentioned this point or not), the localization of the pain to the left temple, were suggestive of a lesion of the higher cerebral centres. I therefore suggested the possibility of a double lesion.

In answer to this letter Dr Barlow wrote me:—"She must have gone down rather badly since I saw her, but your view quite coincides with mine—at least so far—that I thought the tumour was far back and low; and I thought it probable some of the pareses were indirect from pressure rather than involvement; further than that I could not venture. I satisfied myself about the right eye, that there was a little blurring of the edge of the disc and covering over of veins; but I have seen several times now an intercurrent neuritis which has cleared up, so that I am not surprised now that you find *nil* there. I must say, like your-

self, the sum total of the case very strongly seems to me in favour of tumour rather than of softening."

As regards treatment, it was agreed to give iodide of potassium in as large doses as could be satisfactorily borne.

Subsequent Progress of the Case.—For two or three days there was no improvement; in fact, the condition was worse. On 17th October Dr Foulis wrote me:—"Miss A. has been in a very sleepy state since yesterday morning. She can be roused, and will speak to you if you ask questions; but her answers are not correct, wrong words being used. The temperature yesterday was 100° F. This morning the face is flushed. She complains of choking sensations in her throat, and has more difficulty in swallowing. After two or three attempts a violent cough comes on, which gives rise to *agonizing* pain in the *left* of the head.

On the evening of the 18th a blue pill, followed by a saline draught, were prescibed by Dr Foulis; and the free purgation which was thus obtained was followed by marked improvement, which steadily continued.

When next seen by me, on 8th November, she looked very much brighter and better, and was not so pale and anæmic. She had, on the morning of my visit, had a little headache (left-sided and frontal), the first since the improvement began; it was apparently caused by getting up and walking about the room. The difficulty in deglutition had almost completely disappeared; the paralysis and tremor in the right arm and leg were scarcely perceptible; the knee-jerk was now about equal on the two sides; the mental and intellectual faculties were much clearer; the head no longer drooped to the right side; there was no evidence of left-sided facial paralysis; the tongue was slightly furred; the nurse thought the patient was thinner. To continue the iodide; to repeat the blue pill and saline draught occasionally; and to keep very quiet.

The great improvement which had resulted from the free purgation, and perhaps from the use of the iodide, was very remarkable; and although I saw no reason to modify my opinion as to the presence of an intracranial tumour, the continued absence of any left-sided facial paralysis, and the almost complete subsidence of the difficulty of swallowing, threw very grave doubts upon the localization of the lesion which seemed previously indicated; in fact, I now felt disposed to give up the view that the tumour was pressing upon the pons Varolii.

After this date I did not see the patient again during life, but I learned from Dr Foulis that the progress, though interrupted by temporary periods of improvement, was on the whole steadily from bad to worse; until, finally, death took place on March 19th, 1885.

Dr Foulis has very kindly furnished me with the following notes,

which give a much more detailed account of many of the points in the clinical history than I have been able to do:—

"Miss A. consulted me at the end of the year 1883. She complained of severe neuralgia at the back of the head, on the left side. The pain was shooting in character, and seemed to be connected with the great occipital nerve. In appearance she was pale and anæmic. Her expression was emotionless and dull. She did not complain of any want of sensation or muscular power on one side or other. The pupils were equal in size, and responded freely to the action of light. It seemed to me at the time that all her suffering was caused by her very anæmic condition. I therefore prescribed iron and arsenic, with good food and rest.

"I saw her two or three times after this at the beginning of the year, early in January 1884, and, although the anæmia was less, the pain in the region of the great occipital nerve was more intense. I noticed that the glands of the neck on the posterior border of the sterno-mastoid muscle on the left side were enlarged and tender to the touch.

"On the 22nd January 1884 I was sent for at night to see her at her own home. I found her in bed, sleeping so deeply that shouting in her ear and pinching her violently failed to waken her. The limbs that were severely pinched were drawn away slowly, and a slight frown, expressive of pain, was seen on her forehead. The eyelids were nearly closed. The eyeballs were congested, and could be freely touched without causing the eyelids to wink. The pupils were not contracted, and answered the stimulus of light. She was breathing deeply, in a sighing manner; the face was flushed, and covered with perspiration. The temperature in the mouth was over 104° F., while the pulse was 116. On hearing from her friends that she had been suffering from constipation for a few days past, and as there were no one-sided symptoms of muscular or sensory paralysis, I ordered her to have a large dose of castor oil, in the hope that its free action would relieve the cerebral congestion.

"On seeing her next day, I heard that she had slept deeply for twenty hours, and that as soon as the castor oil had acted freely she began to revive, and shortly afterwards became quite conscious, but had no recollection whatever of what had occurred the previous night. For some weeks after this I saw her frequently. The pain in the back and left side of the head never left her, day or night. She was sometimes sick and vomited her food. There was marked constipation at all times, which necessitated the use of aperient medicine. At times the pain in the head was agonizing, but was always relieved after a free action of the bowels. It was noticed that she now occasionally made use of wrong words in conversation, and she introduced persons and ideas quite unconnected with the subject of conversation; of this she was quite

unconscious. Her friends now reported to me that my patient had fallen asleep in a bath in the house of a relative, but this sleep did not last for more than a couple of hours. About this time it was observed that her right arm was deficient in muscular power, and that in walking she fell away to the right side. She could not squeeze so well with the right as with the left hand, and on raising a cup of tea to her mouth the cup shook very much and was in danger of falling. She joined in conversation on all subjects, but frequently made mistakes without being aware of it. At times there was a difficulty in swallowing, and the bladder sometimes failed to retain the urine.

"On the 5th May she had another very extraordinary sleeping fit, accompanied by fever and profuse perspiration. This comatose state lasted for quite twenty hours, and was put an end to by a large dose of aperient medicine as already described. After this, for some weeks, she decidedly improved in every respect; so much so, that she was able to accompany her friends to Moffat at the beginning of summer. On the arrival of the train at Moffat she appeared to be very drowsy, and as soon as she reached her house she fell into a deep sleep, from which she did not wake for twenty-four hours. She was seen by a local medical gentleman, who gave her the usual large dose of aperient, and it was followed by complete relief from all anxious symptoms, as on former occasions. She remained several weeks at Moffat, during which time she had two or three sleeping fits, as above described. In general condition she was not worse when she returned to Edinburgh at the end of summer. The pain in her head still continued very severe, and it was quite evident that she was duller in her mental condition. Her expression was now decidedly emotionless, but she was fairly well nourished, and her anæmia had quite disappeared.

"Towards the end of July she was seen by Dr Barlow in consultation with me. He expressed the opinion that there was a tumour growing in the brain, but did not speak definitely as to its localization. In the middle of October she was seen by Dr Byrom Bramwell with me in consultation, who examined her very carefully, and as the result of that examination he diagnosed a tumour, and stated the grounds on which he localized the new growth. To relieve the pain in the head it was decided to give her ten-grain doses of potassium iodide three times daily. This was followed with such relief to the pain in the head and to all other urgent symptoms that her friends began to hope that she was going to get better; but at the end of three weeks' trial we were obliged to give up the potassium iodide as it produced such general weakness, and it at last lost its power in relieving the headache. In one of her deep sleeping fits, I found that a blue pill followed by a saline purge in the morning gave her marked relief from all her suffering, and this treatment was continued for some weeks, but, like everything else, it at last failed, and all the symptoms got

gradually worse. The pain in the back and left side of the head was now at times quite agonizing. She frequently vomited her food. She had great difficulty in masticating and swallowing. Violent fits of coughing came on as the result of crumbs of bread or other food going down the wrong way. She suffered intense pain in her head during the fits of coughing. She was now very apathetic and listless. There was decided motor and sensory paralysis on the right side, and her urine often came away involuntarily. A bed-sore appeared on her back, and she was now placed in bed on a water-bed. She was seen by another gentleman in consultation with me, who also diagnosed a cerebral tumour, but localized it differently from the other consultants. It was now necessary to feed her on liquid food, as she could not masticate. She could speak in answer to questions, but with great difficulty, and only in a whisper. Her pulse was generally about 96, while the temperature reached about 100° F. at night-time.

"At the beginning of the year 1885 she improved in general condition so much that she was able to get out of bed, and sat in a chair, and actually received some of her friends to tea. After being in such a serious condition in December 1884, it seemed almost a miracle that she could leave her bed in January 1885. With a little support she could rise out of her chair and walk a few yards, though it was quite evident there was great muscular and sensory weakness in the right limbs. Her powers of conversation greatly improved, so that she could carry on a little chat with those around her.

"This improvement, however, did not last for more than a few days, and as the urgent symptoms gradually returned, she was replaced in bed on the water-bed. From the end of January on to the 19th March, when she died, her condition may be simply described as one of right-sided paralysis with aphasia.

"About a month before she died the pupils of her eyes were moderately dilated, and did not respond in the least to the stimulus of a strong light. Her power of mastication was now quite gone, though she gulped down liquids after they were poured into her buccal cavity. Urine and fæces escaped involuntarily. She gradually became deeply unconscious to everything around her, and day by day, as the temperature and pulse rose, she became weaker and weaker, till she died exhausted on the 19th March. For the last week or ten days the temperature varied from 100° F. to 104° F., and her dying hours were of extraordinary duration."

The post-mortem examination was made on March 20th; the head only was examined.

The scalp, skull-cap, and outer surface of the dura were natural; the internal surface of the dura was adherent over the left frontal lobe, which was markedly swollen and considerably larger than the right. There was no appearance of any general increase of the

intracranial pressure—no flattening of the convolutions, no effacement of the sulci; in fact, the convolutions of the brain generally were rather atrophied than flattened, the sulci wide, and the subarachnoid fluid in some excess.

On cutting up the brain the greater portion of the left frontal lobe, or to speak more precisely, the anterior two-thirds of the first and second, and the anterior half of the third left frontal convolutions, with the associated portions of the subjacent white matter, were completely destroyed by a new growth; while the posterior third of the first and second, and the greater part of the posterior half of the third left frontal convolutions, were softened, œdematous, and, to some extent, infiltrated by the lesion (see Fig. 1). The greater portion of the tumour consisted of a soft glioma, but embedded in its posterior end, and projecting for some distance behind it, there was a cyst as large as an average size hen's egg.

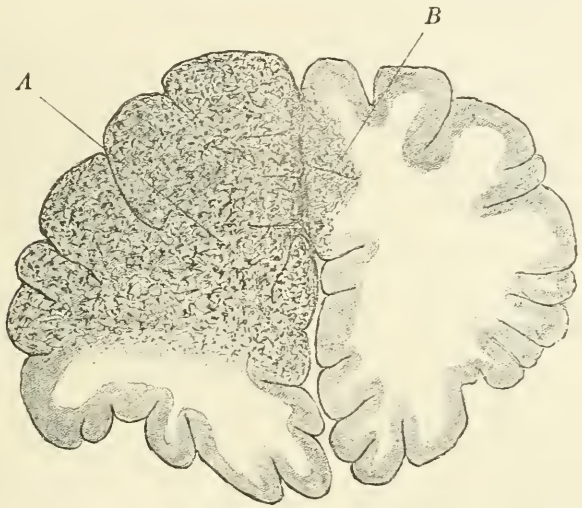


Fig 1.—*Transverse Vertical Section through the Frontal Lobes in the case of Miss A., showing the position of the lesion.* Drawn from memory.

A points to the lesion in the left frontal lobe, and *B* to a portion of the right frontal lobe affected by the direct extension of the tumour.

This cyst, which contained a colourless but slightly turbid fluid, was bounded above and in front by the gliomatous tissue of the tumour, below and behind by the anterior cornu of the lateral ventricle, and behind by the white matter of the centrum ovale.

The posterior end of the cyst was in close contact with the lenticular nucleus and head of the corpus striatum, and must have been in a position to have exerted pressure upon those fibres of the centrum ovale which pass downwards under the name of the motor portion of the internal capsule. The wall of the cyst was formed by a tough but transparent membrane, which was the only

structure which divided the cavity of the cyst from the cavity of the lateral ventricle; in this semi-transparent partition a most beautiful network of bloodvessels was seen.

Several recent hæmorrhages, the largest about the size of a large walnut, were present in different parts of the tumour; and the pigmented remains of other hæmorrhages of old date were visible, both to the naked eye and on subsequent microscopical examination, in other parts of the new growth.

The enlarged and diseased left frontal lobe bulged across the middle line, and at one spot where it impinged upon the right frontal lobe (see B, Fig. 1), the tissue of that (the right) lobe was diseased, the result without doubt of a process of direct extension or auto-inoculation.

The other portions of the brain were free from disease. The optic nerves were to the naked eye perfectly normal.

On microscopical examination, the tumour was found to be a glioma, or perhaps more correctly a glio-sarcoma.

The cells were for the most part round or oval, and arranged in dense masses (see Figs. 7, 11, 14). In some parts, more especially in half-cleared-up carmine or picro-carmine preparations, and in sections stained with picro-carmine, teased out, and mounted in Farrant's solution, the glial threads or fibres were well shown (see Fig. 2). In those portions of the tumour into which recent

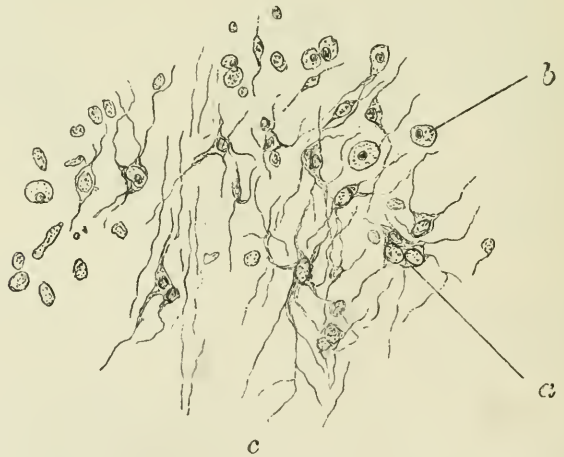


Fig. 2.—*Camera Lucida Drawing of a Microscopical Section of a portion of the Cerebral Tumour in the case of Miss A., showing Gliomatous and Sarcomatous Cells and Fine Fibres. Stained with Picro-carmine, half-cleared with Methylated Spirit and Oil of Cloves, and mounted in Zylol Balsam. (Magnified—Hartnack, oc, 3. obj. 8.)*

a, Glial cell containing three nuclei; *b*, round sarcomatous cell containing nucleus; *c*, fine glial fibres.

hæmorrhages had occurred, large round cells, which appeared to be distended with clear colourless contents, which did not stain pink with picro-carmine, were found. Some of these large swollen

cells contained a single small round nucleus, others contained red blood corpuscles which they had apparently digested (see Fig. 3). The impression left upon my mind was that the large swollen cells with colourless non-staining contents had previously contained red blood corpuscles, which had become fused into a single uniform mass.

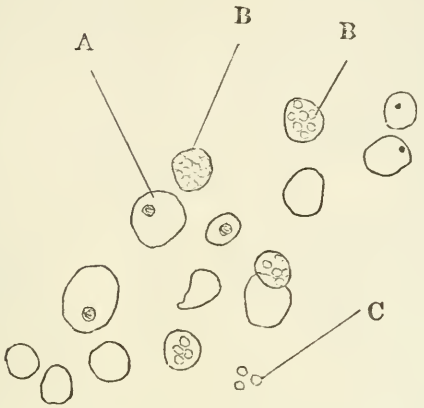


FIG. 3.—*Camera Lucida Drawing of a Microscopical Section of a portion of the Cerebral Tumour in the case of Miss A., showing large round Transparent Cells, some of which contain a single small Nucleus (? Nucleolus), others Red Blood Globules. These Cells lay in the midst of Extravasated Red Blood Corpuscles, which have been omitted from the Drawing. Stained with Picro-carmine, half-cleared with Methylated Spirit and Oil of Cloves, and mounted in Zylol Balsam. (Magnified—Hartnack, oc. 3, obj. 8.)*

A, Large cell containing a single nucleus; B, B, cells containing red blood corpuscles; C, red blood globules.

The tumour tissue was most vascular—indeed, in some places it appeared to consist of a dense network of minute vessels, in the meshes of which the glial cells were situated. Aneurismal dilatations were, in many places, seen on the minute vessels (see Fig. 4). In places, therefore, the structure of the tumour corresponded to the condition which has been termed a telangiectatic glioma.

In the midst of some of the large masses of extravasated red blood corpuscles, which were situated in different parts of the tumour, enormous crystals were situated (see Fig. 5). These crystals were of a pale yellow colour; they were seen both in preparations mounted in Farrant's solution, and in sections which had been treated with absolute alcohol and oil of cloves, and mounted in balsam. I am unable to pronounce an opinion as to their exact nature, for I have never seen anything resembling them before. I feel convinced that they were not artificial (post-mortem) productions, the result of the reagents employed in hardening and mounting the sections. Possibly they were derivatives of the red blood globules in the midst of which they lay; but whether they existed during life, or were produced during the death agony, or even after death, I feel unable to decide.

Surrounding the masses of extravasated blood in which the crystals were embedded, and in the midst of some of the other extravasations in which there were no crystals, large irregularly

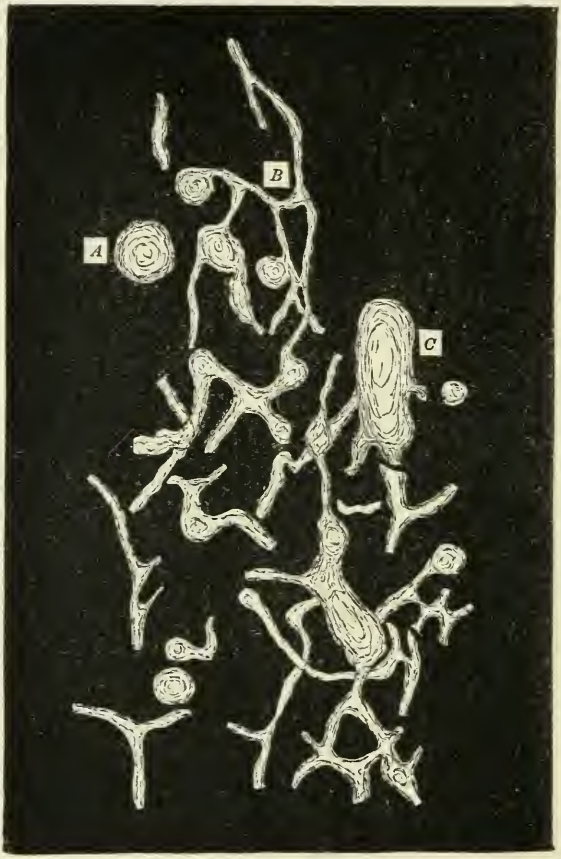


FIG. 4.—*Camera Lucida Drawing of a Microscopic Section of a portion of the Cerebral Tumour in the case of Miss A., showing Aneurismal Dilatation of the Minute Vessels. Stained with Picro-carmine, cleared with absolute Alcohol and Oil of Cloves, and mounted in Zylol Balsam. (Magnified—Hartnack, oc. 3, obj. 8, and drawing reduced from 8 to 4 inches.)*

A, Transversely divided vessel; *B*, vessel with numerous aneurismal dilatations; *C*, large aneurismal dilatation. The tissue of the tumour in the midst of which the vessels lie, and many of the vessels themselves, have been omitted from the drawing.

shaped masses, composed of a highly refractive, transparent, and structureless material, were situated. These masses were for the most part of the same pale yellow colour as the large crystals, but some of them stained pink with picro-carmine. These irregular, structureless, colloid-like masses were probably like the crystals, derivatives of the extravasated red blood corpuscles. Further, I am disposed to think, after careful examination of many different

preparations, that they represented a material in an intermediate stage between the extravasated red blood corpuscles and the hyaline material which was present in the walls of many of the bloodvessels of the tumour, and to which I will presently refer. The crystals, the irregularly shaped, transparent, yellow masses, and the hyaline material in the walls of the vessels, were present

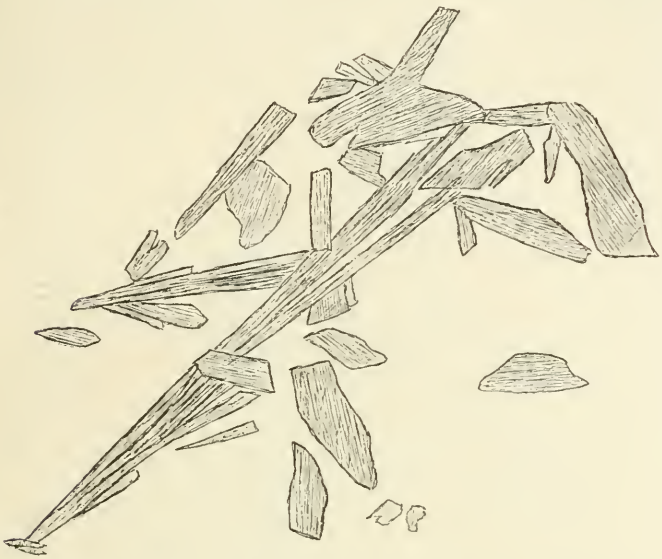


FIG. 5.—*Camera Lucida Drawing of a Microscopical Section of a portion of the Cerebral Tumour in the case of Miss A., showing the Large Crystals described in the Text. The Blood Corpuscles, in the midst of which the Crystals lie, are not shown in the drawing, and could hardly be individually distinguished with this Magnifying Power—Hartnack, oc. 3, obj. 3, and drawing reduced from* 4 *to* 3½ *inches. The Preparation was stained with Picro-carmine and mounted in Farrant's Solution.*

in such quantities that it is hardly possible, I think, to suppose that they were derivatives of the extravasated *white* blood corpuscles. Possibly, however, the hyaline material which was present in such abundance in the walls of the bloodvessels, both in this case and in Dr Leslie's case of glio-sarcomatous tumour, which will be subsequently reported, may have been a derivative—a product so to speak—of the glial cells. But be that as it may, it is important to note that in these two cases in which the hyaline degeneration of the vessels was so extensive, the structure of the two tumours was identically the same; both were glio-sarcomatous tumours; in both there was a large cyst connected with the tumour; in both the tumour tissue contained an enormous number of minute vessels; and in both there was evidence of extensive blood extravasations.

In many parts of the tumour highly refractive, homogeneous masses, composed of hyaline material, were seen. These hyaline masses were in some places most numerous; many of them were

small, others very large; the large masses were almost invariably stained pink when treated with picro-carmine. Some of the small masses did not take on the pink stain, but remained unstained, or were stained of a pale yellow or greenish-yellow colour; in fact, they closely resembled little drops or beads of yellowish-green glass. The large hyaline masses were much more

FIG. 6.—*Camera Lucida Drawing of a Microscopical Section through a portion of the Cerebral Tumour in the case of Miss A., showing numerous Hyaline Lumps. The Tumour Tissue, in the midst of which the Hyaline Masses lie, has not been represented, for, under this magnifying power the Individual Cells of the Tumour could not be distinguished. Stained with Picro-carmine, cleared with absolute Alcohol and Oil of Cloves, and mounted in Zylol Balsam. (Magnified—Hartnack, oc. 2, obj. 3, and drawing reduced from 6½ to 4½ inches.)*

A, B, point to two of the hyaloid lumps, which are seen to be concentrically lined, and are in reality hyaline masses surrounding bloodvessels.

highly stained in sections which were treated with alcohol and oil of cloves and mounted in balsam than in sections mounted in Farrant's solution.

In completely cleared-up carmine and picro-carmine preparation

(*i.e.*, sections which had been treated with absolute alcohol), the large hyaline masses were concentrically lined; in sections half-cleared-up (*i.e.*, treated with methylated spirit), and still better in preparations mounted in Farrant's solution, one or more minute vessels, which were, as a rule, transversely divided, could be seen in the midst of the hyaline material. The concentric appearance seen in completely cleared-up preparations was evidently the result of the shrinking produced in the hyaline material by the absolute alcohol, for the concentric markings were less marked in half-cleared-up preparations, and not seen in sections mounted in Farrant's solution.

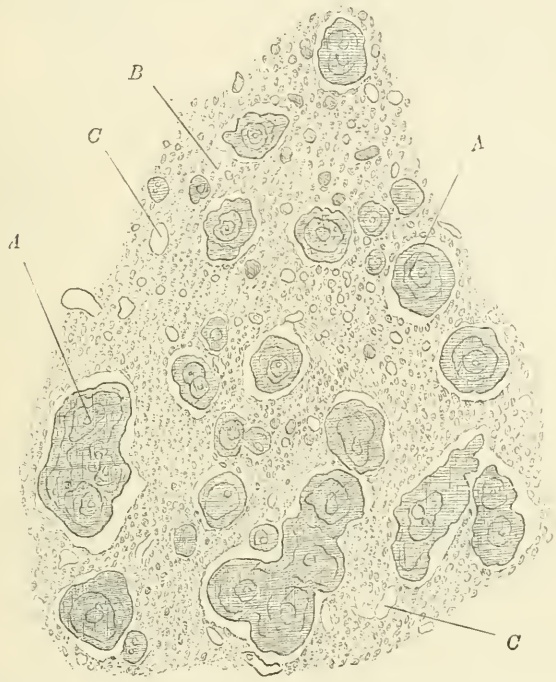


FIG. 7.—*Camera Lucida Drawing of a Microscopical Section of a portion of the Cerebral Tumour in the case of Miss A., showing large Hyaline Masses lying in the midst of Sarcomatous Tissue. Stained with Picro-carmine, cleared with absolute Alcohol and Oil of Cloves, and mounted in Zylol Balsam. (Magnified—Hartnack, oc. 3, obj. 7, and drawing reduced from 5½ to 3½ inches.)*

A, A, Large hyaline masses, concentrically lined, and containing minute bloodvessels; *B*, sarcomatous tissue; *C, C*, small vessels in the tumour, the walls of which are healthy.

The small hyaline masses were in some cases connected with vessels, but in many places this connexion was not apparent, and the little hyaline masses then appeared to be simply scattered here and there in the midst of the tissue of the tumour. The appearances just described are well seen in Figs. 6, 7, and 8. In 6 and 8 the tumour tissue is not represented, but in Fig. 7 the comparative size of the glial cells and hyaline lumps is seen.

In other sections long hyaline masses evidently surrounding minute vessels were seen (see Figs. 9 and 10). One of these

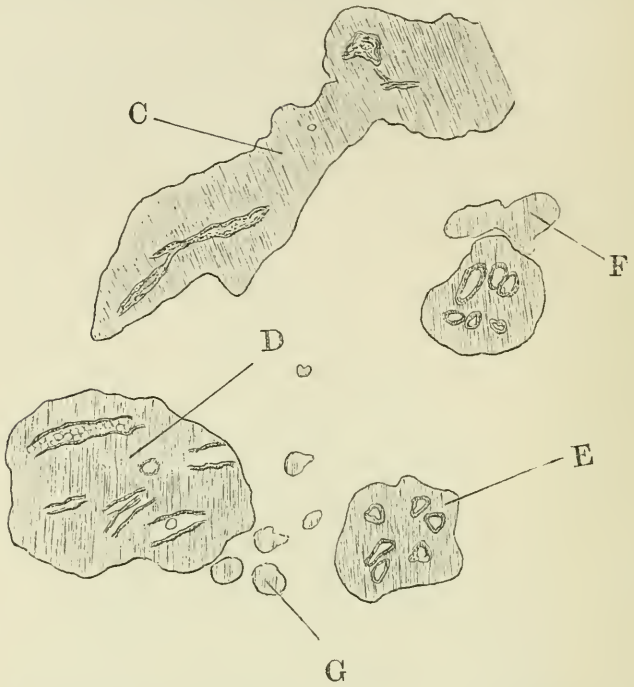


FIG. 8.—*Camera Lucida Drawing of Hyaline Masses from the Cerebral Tumour in the case of Miss A., showing numerous small Vessels in their Interior. Stained with Picro-carmine, and mounted in Farrant's Solution.* (*Magnified—Hartnack, oc.* 3, *obj.* 8.)

C, D, Hyaline lumps, containing minute vessels longitudinally and transversely divided; E, hyaloid lump, containing six minute vessels transversely divided; F and G, large and small hyaline lumps, in which there are no vessels.

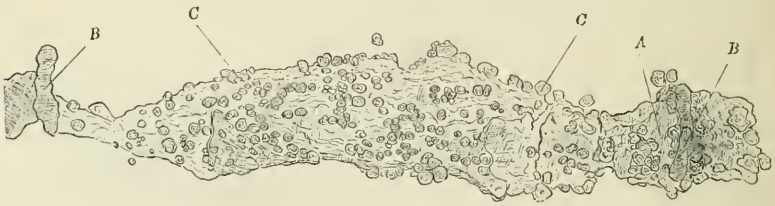


FIG. 9.—*Camera Lucida Drawing of a Bloodvessel from the Cerebral Tumour in the case of Miss A., showing Enormous Thickening, due to Hyaline Infiltration of its Wall, and numerous small Hyaline Globules, which appear to have exuded from the Outer Surface of the Hyaline Sheath. Stained with Picro-carmine, cleared with absolute Alcohol and Oil of Cloves, and mounted in Zylol Balsam.* (*Magnified—Hartnack, oc.* 3, *obj.* 8, *and drawing reduced from* 8 *to* 3 *inches.*)

A, Hyaline sheath; *B, B*, large, and *C, C*, small masses of hyaline material adhering to the outer surface of the hyaline sheath. The small hyaline masses are concentrically lined.

hyaline masses was so cut that the minute vessel in its interior could be clearly seen; in this instance the hyaline mass seemed to

form a sheath around the vessel; between the vessel and the hyaline cylinder some delicate spindle cells and connective tissue fibres were situated.

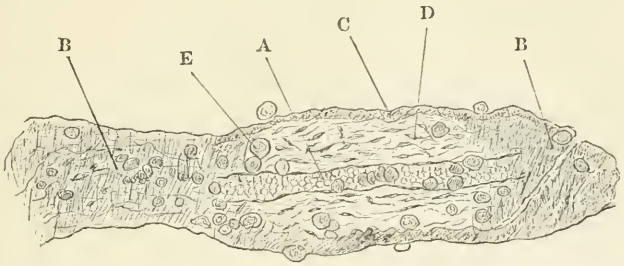


FIG. 10.—*Camera Lucida Drawing of a Minute Vessel and the Hyaline Sheath which surrounds it, from the Cerebral Tumour in the case of Miss A. The Hyaline Sheath is so cut that the Minute Vessel in its Interior is distinctly seen. Between the Vessel and the Hyaline Sheath some delicate Spindle Cells and Fibres are situated. Stained with Picro-carmine, cleared with absolute Alcohol and Oil of Cloves, and mounted in Zylol Balsam. (Magnified—Hartnack, oc. 3, obj. 8, and drawing reduced from 4½ to 3¼ inches.)*

A, Vessel containing red blood corpuscles and some small round hyaline lumps; B, hyaline sheath surrounding the vessel; at B′ the hyaline sheath is cracked across; C, cut edge of hyaline sheath; D, spindle cells between the sheath and its contained vessel; E, hyaline globules which are adhering to, and in places appear to have exuded from, the hyaline sheath.

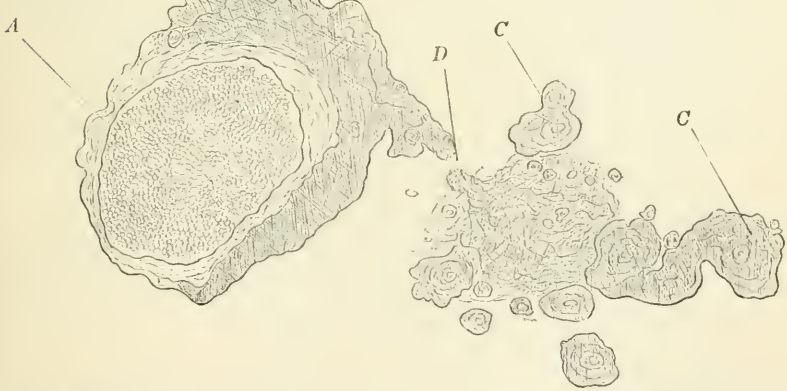


FIG. 11.—*Camera Lucida Drawing of a Section through a portion of the Cerebral Tumour in the case of Miss A., showing a Bloodvessel, the Wall of which is partly infiltrated with Hyaline Material, which is apparently continuous with Hyaline Masses in the Tissue of the Tumour surrounding it. Stained with Picro-carmine, cleared with absolute Alcohol and Oil of Cloves, and mounted in Zylol Balsam. (Magnified—Hartnack, oc. 3, obj. 8, and drawing reduced from 6 to 4 inches.)*

A, Healthy portion of vessel wall; *B*, Hyaline mass in vessel wall; *C, C*, hyaline lumps in surrounding tissue; *D*, the junction of the hyaline mass in wall of vessel and in surrounding tissue; the continuity has apparently been broken in the process of preparation. The sarcomatous tissue in which the vessel lies has been omitted from the drawing.

Adhering to the outer surface of these long hyaline sheaths were numerous small round or oval hyaline masses, which looked

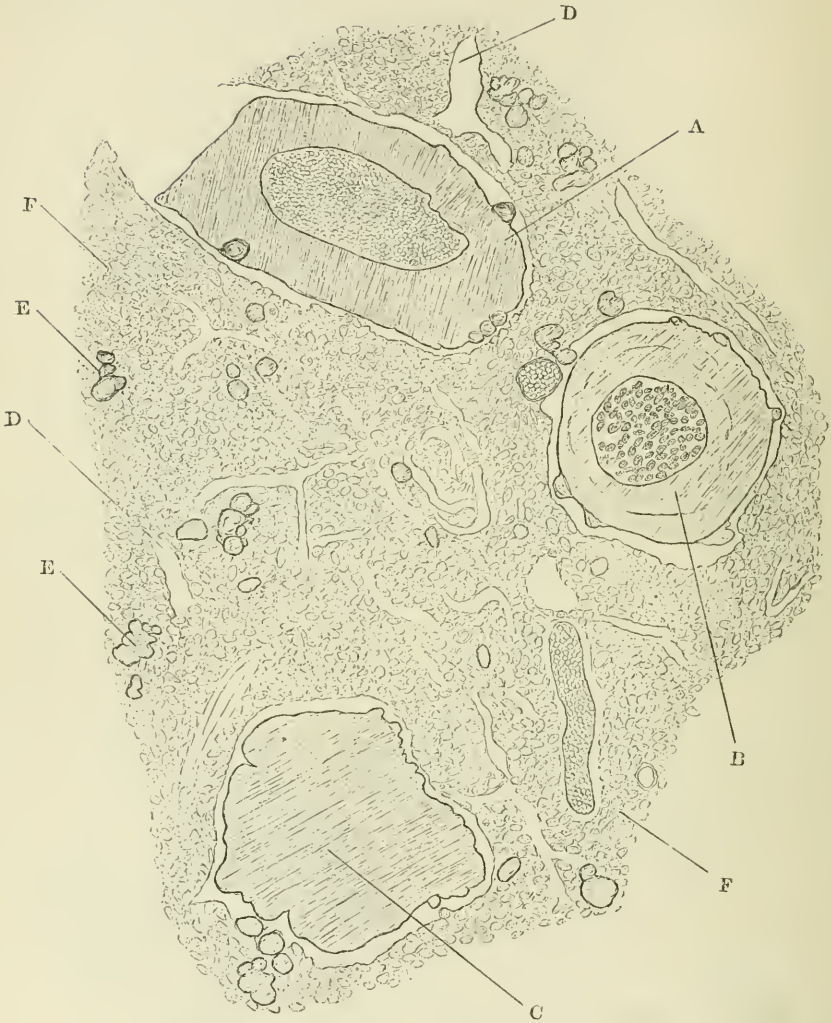


FIG. 12.—*Camera Lucida Drawing of a Microscopical Section of a portion of the Tumour in the case of Miss A., showing three Vessels, the Walls of which are enormously thickened and infiltrated with Hyaline Material. Stained with Picro-carmine, mounted in Farrant's Solution. (Magnified—Hartnack, oc. 3, obj. 8, and drawing reduced from 7 to 4¼ inches.)*

A, Vessel containing blood corpuscles—its walls are enormously thickened and infiltrated with hyaline material; B, vessel with walls affected in the same manner as A, but filled with sarcomatous cells, forming, as it were, a sarcomatous infarction; C, large mass of hyaline material, which seems to represent a former bloodvessel; D, D, small vessels in the midst of the sarcomatous tissue; E, E, small hyaline lumps in the midst of the sarcomatous tissue; F, F, point to the sarcomatous tissue, which is composed at this part of round and oval cells.

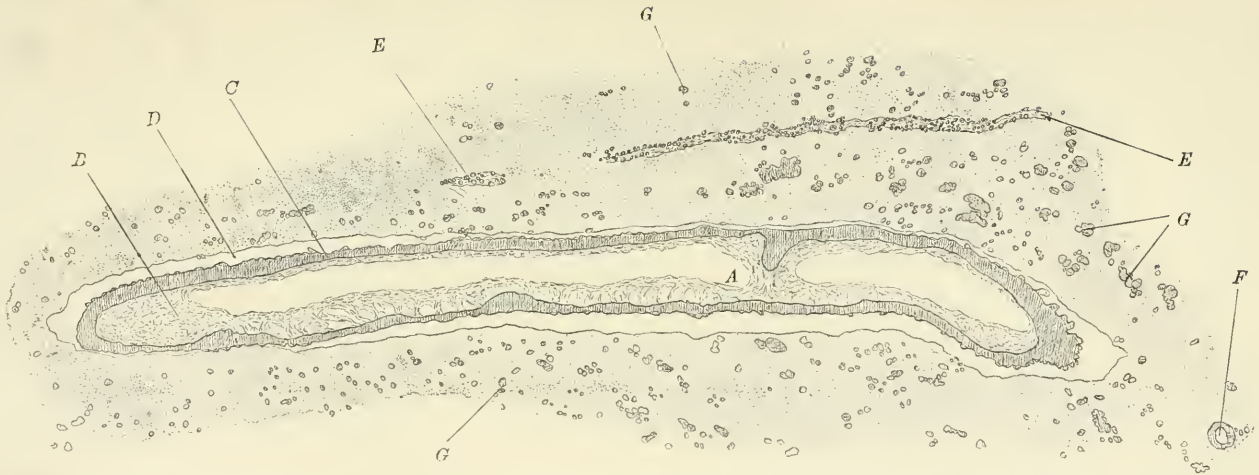


FIG. 13.—*Camera Lucida Drawing of a Microscopical Section through a portion of the Cerebral Tumour in the case of Miss A., showing a large Vessel in Longitudinal Section; the Coats of the Vessel are infiltrated with Hyaline Material, and its Canal is partly obstructed by an organizing Thrombus; numerous small Hyaline Lumps are seen in the surrounding Tissue—some of them appear to be free, others adhering to Minute Vessels. Stained with Picro-carmine, cleared with absolute Alcohol and Oil of Cloves, and mounted in Zylol Balsam. (Magnified—Hartnack, oc. 3, obj. 3, and drawing reduced from* 11 *to* 6½ *inches.)*

The letter *A* is placed in the interior of the vessel, near a point where its canal is bridged across by a mass of hyaline material and by a portion of the organized thrombus (*B*); *C*, hyaline material in walls of vessel; *D*, space (? lymphatic space) surrounding the bloodvessel; *E, E*, minute vessel, seen longitudinally, surrounded by numerous small hyaline masses; *F*, small vessel, transversely divided, the coats of which are infiltrated with hyaline material; *G, G, G*, small hyaline lumps in the tissue of the tumour, the minute structure of which cannot be seen under this low magnifying power.

as if they had exuded in the form of drops from the main mass of hyaline substance. The same droplike appearance was also seen in transverse sections of vessels affected with the hyaline change (see Figs. 12 and 13).

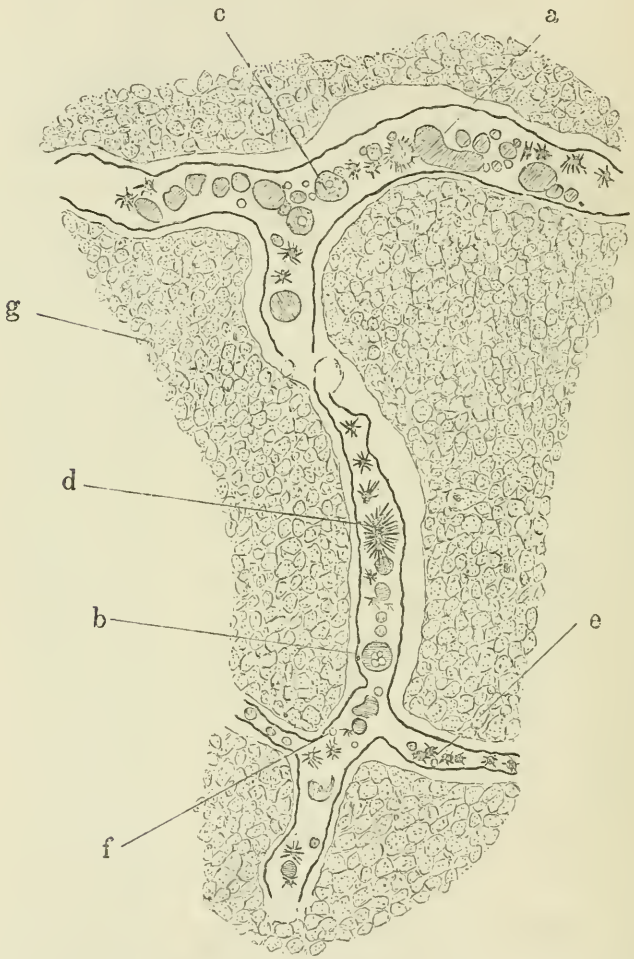


FIG. 14.—*Camera Lucida Drawing of a Section through the Cerebral Tumour in the case of Miss A., showing a Bloodvessel surrounded by Sarcomatous Tissue; in the interior of the Vessel there are numerous masses of Hyaline Material, some of them contain small round Bodies (Vacuoles, or Red Blood Corpuscles), others are bristled with Acicular Crystals. Stained with Picro-carmine, cleared with absolute Alcohol and Oil of Cloves, and mounted in Zylol Balsam. (Magnified—Hartnack, oc. 3, obj. 8, and drawing reduced from 5½ to 5 inches.)*

a, Large irregularly shaped hyaline mass, b and c, round hyaline masses containing small, clear, round bodies (? vacuoles or red blood corpuscles); d, large oval hyaline mass, bristled with acicular crystals; e, small round hyaline masses, studded with acicular crystals; f, red blood corpuscle; g, sarcomatous tissue, composed of round and oval cells. There were numerous minute bloodvessels amongst the sarcomatous cells they are not shown in the drawing.

The walls of many of the large vessels contained in the tumour were also infiltrated with hyaline material (see Figs. 11, 12, and 13). In some instances (as in Fig. 11), the hyaline infiltration was limited to one side of the vascular wall, and in many cases the hyaline material in the vascular wall appeared to be continuous with masses of hyaline material in the tissue of the tumour surrounding the affected vessel.

Some of the large vessels affected with the hyaline change were filled with blood corpuscles (see A, Fig. 12); in one instance (B, Fig. 12) the vessel contained glial cells—an infarction or thrombosis composed of cells identical in structure, though not quite so large in size, as the cells forming the adjacent portion of the tumour. In several instances, vessels, the walls of which were infiltrated with hyaline material, were partly filled up by an organizing thrombus (see Fig. 13).

In many different sections hyaline lumps, for the most part round or oval, and many times larger than red blood corpuscles, were seen in the interior of vessels, the walls of which were healthy. Some of these masses contained round, clear, colourless globules in their interior—probably vacuoles, but possibly red blood globules. The surface of the hyaline masses within the bloodvessels was in many instances bristled with fine acicular crystals; these crystals, which were stained in picro-carmine preparations of the same pink colour as the hyaline material, had probably been formed after death or during the death agony. In Fig. 14 a remarkable illustration of these intravascular, hyaline masses and crystals is faithfully represented; and it is to be observed that in the portion of the tumour surrounding this vessel no hyaline masses are to be seen.

Remarks.—This case presents several points of great clinical and pathological interest.

Diagnosis.—It could hardly, I think, be doubted, that the condition of the patient when I first saw her, and the whole previous history of the case, were indicative of an organic cerebral lesion; while the history of headache and vomiting, the pseudo-apoplectic attacks, the one-sided paralysis and tremor which varied so remarkably from time to time, and the temporary and unilateral optic neuritis observed by Dr Barlow, together with the absence of any of the ordinary causes of cerebral softening, were strongly suggestive of an intracranial tumour.

The reason which induced me, when I first saw the patient, to think that the tumour was pressing upon the pons Varolii have already been detailed. The appearances found at the autopsy showed no cause for the left-sided facial paralysis, nor of the extreme difficulty of swallowing which was such a marked symptom at the time of my first visit.

On reviewing the whole history and progress of the case, I am disposed to think that the character of the intellectual deteriora-

tion (the marked loss of the power of attention and of mental concentration), the falling of the head to the right, and the defects in speech (use of wrong words, etc.), which might be called a minor degree of aphasia, should have enabled us to localize the lesion in the left frontal lobe. It must, however, be remembered that many of the symptoms to which I have just referred were by no means prominently marked, that almost all of them were temporary and evanescent (present at times, absent at others), and that it seemed difficult to explain the unilateral paralysis and voluntary tremor, and to account for the remarkable variability of these symptoms (paralysis and tremor) by a tumour in the frontal lobe.[1]

Further, a careful review of the symptoms in the light of the conditions found at the post-mortem leads me to think that the exact pathological nature of the tumour might perhaps have been correctly surmised.

The coarse tremor, absent while the limbs were at rest, but present on voluntary movement (granting that it was due to organic and not to functional—hysterical—disease), was indicative either of cerebro-spinal sclerosis or of pressure on the motor conducting fibres.

The facts that the paralysis and tremor varied greatly in degree from time to time, and had on more than one occasion completely disappeared, excluded cerebro-spinal sclerosis (indeed that condition was never suspected, for the symptoms, taken as a whole, were in no way indicative of its presence), and ought, perhaps, to have suggested the presence of a cyst, or at all events of fluid pressure.[2]

[1] In the *British Medical Journal*, 3rd March 1876, and in the *Edinburgh Medical Journal*, December 1878, I have reported cases in which tumours with cysts were found in the frontal lobes. In both cases the mental symptoms were very prominent, and in both there were sudden but temporary attacks of hemiplegia without loss of consciousness, and without convulsions. In one case there was also some loss of power on the same side (arm and leg) as the lesion. These two cases have several points in common with the one which I am at present describing.

[2] The pressure of a cerebellar tumour on the pons or medulla is a common cause of this form of voluntary tremor. For the past ten years I have been familiar with cases of this kind, and have explained the tremor by the pressure of the tumour upon the motor conducting fibres in the pons Varolii or medulla. I have also seen two cases in which a lesion, presumably in the neighbourhood of the lenticular nucleus (for the diagnosis was not confirmed by post-mortem examination), has been attended with the same form of voluntary tremor, the result, I suppose, of pressure on the motor strands of the internal capsule. But in these cases there were marked sensory disturbances, the result no doubt of pressure on the sensory fibres of the internal capsule. I have met with no case in which a tumour of the frontal lobe by pressing upon the motor fibres of the internal capsule has caused tremor of this kind—unless such was the position of the lesion in a case which I saw some two years ago with Dr Struthers of Leith. In many respects that case bore such a close resemblance to the one (case of Miss A.) which I am now describing, that although there was no post-mortem, I will on some future occasion record its chief details.

Granting, then, as might perhaps have been conjectured, that the intermittent character of the paralysis and tremor could only have resulted from the pressure of fluid, the tension of which varied in degree from time to time, it might, I think, further have been supposed that the cause of the pressure was the presence of a cyst. The variations in the degree of pressure, as evidenced by the paralysis and tremor, were so rapid and extensive that an aneurism—another possible cause of intermittent pressure—might, I think, have been definitely excluded.

Now, if we exclude parasitic cysts (hydatids and cysticercus cellulose), which, in such a case as this, might probably be excluded—*firstly*, because of their extreme rarity; and, *secondly*, because there was no evidence of similar disease in any other part of the body—a cyst of sufficient size to produce the symptoms which have been described must in all probability have been the result of an ordinary hæmorrhagic apoplexy or of a new growth (glioma with hæmorrhage and cyst formation).

Further, ordinary cerebral hæmorrhage (hæmorrhagic apoplexy *par excellence*) might, I think, have been definitely excluded as the cause of the supposed cyst—*firstly*, because there was no evidence of any of the conditions which are usually associated with ordinary cerebral hæmorrhage (cardiac, vascular, or kidney disease); *secondly*, because the symptoms, both individually (headache, vomiting, pseudo-apoplectic attacks) and as a whole, and the whole progress and course of the case, were strongly in favour of a tumour rather than of softening, the result of hæmorrhagic apoplexy. In particular, the numerous pseudo-apoplectic attacks are easily explained by repeated small hæmorrhages in the substance of a tumour, and the disturbance of the intracranial pressure produced thereby, but would have been difficult or impossible to explain on the supposition that the supposed cyst had resulted from ordinary hæmorrhagic apoplexy. It is hardly possible to imagine so many separate hæmorrhagic extravasations, all of small size, all recovered from within a few hours, all outside the ordinary lenticulo-striate region, for none of the hæmorrhages were followed by motor or sensory paralysis.

The fact that sudden attacks of hemiplegia, rapidly passing off, were observed in two other cases of cyst in the frontal lobes, has already been referred to.

Reviewing, then, the symptoms which were observed during life in the light of the appearances found at the post-mortem, and taking into account the other two cases of tumour with cyst in the frontal lobe which I have reported, I am disposed to think that the position and exact pathological nature of the lesion might perhaps have been correctly surmised.

The effect of free purgation on the pseudo-apoplectic attacks is another point of great clinical interest, and is, I think, satisfactorily explained by supposing that the drain of water from the bowel by

reducing the general blood pressure, produced absorption of fluid, which had been effused into the tissues of the tumour, while it, perhaps, at the same time reduced the tension of the fluid in the cyst; and that in one or other or both of these ways the increased tension within the cranium was reduced, and relief afforded.

The condition of the deep reflexes is a point of considerable interest. At the time of my first visit there was marked paralysis of the right arm and leg, but the knee-jerk was diminished instead of increased, as it usually is in cases of cerebral hemiplegia. When I next saw the patient the hemiplegia had almost completely disappeared, and the knee-jerk had reappeared, and was, in fact, equal to that on the left (non-paralyzed) side.

This fact would seem to show that where paralysis results from *simple* pressure upon motor conducting fibres (*i.e.*, where there are no irritative changes in the conducting fibres as the result of that pressure) the knee-jerk is diminished rather than exaggerated—an important clinical indication of the cause of the paralysis, if it should be verified by future observations.

Further, if the effect of simple pressure is to produce diminution of the deep reflex movements, the exaggeration of the deep reflexes, which is present in ordinary hemiplegia, must evidently depend upon a process of irritation passing down the pyramidal tract to the multipolar nerve cells of the spinal cord, and not upon the simple arrest or shutting off of the cerebral control—the other theory which has been advanced to account for the exaggeration in that condition.

The character of the mental deterioration which was present in this case, and the *marked way in which the head tended to fall to the right side* (the side opposite the lesion), confirm Ferrier's observations on lesions of the frontal lobes.

The exact duration of the lesion must be a matter of speculation. It can hardly be imagined that the cerebral attack which was said to be due to "water on the head," whatever that term may mean, and which occurred at the age of twelve (thirty-five years before the patient's death), had anything to do with the condition.

Whether the well-marked cerebral attack (coma, rigidity, and speechlessness, lasting for five days) which occurred at the age of thirty-five was due to the lesion which was found at the autopsy it is impossible to say. It must, however, be observed that after this attack a distinct and permanent change was observed in the patient's mental condition; presumably, therefore, there had been some lesion which had resulted in permanent damage. It is not unreasonable to suppose that the cause of this attack was a hæmorrhage into the left frontal lobe, and that the cyst resulted from that hæmorrhage.

The condition of the optic nerve and retina.—The fact, that in this case, in which (*a*), the lesion was distinctly irritative in character

(evidenced by its microscopical characters, by the adhesion of the dura mater over the tumour, and by the direct extension—auto-inoculation—of the new growth from the surface of one to that of the other frontal lobe), and in which (*b*) there was no evidence of increased intracranial pressure at the autopsy, there was only a passing neuritis in one fundus, supports, I think, the view which I have always held, that increased intracranial pressure is a most important factor in the production of the double optic neuritis which is so frequently observed in connexion with intracranial tumours. It must, however, be remembered that Dr Hughlings Jackson has advanced the fact that, in those rare cases in which the neuritis is unilateral, it is present, as it was in this case, on the opposite side to the cerebral lesion, as an argument against the increased intracranial pressure theory.

Points of pathological interest.—The extreme vascularity of the tumour, the evidence of many separate small hæmorrhages in the tissue of the tumour, the frequent relationship of cysts with gliomatous tumours, the direct extension of the tumour by a process of auto-inoculation from the surface of the left to that of the right frontal lobe, the glio-sarcomatous infarction which was present in one of the vessels of the tumour (see Fig. 11), and the peculiar character of the large crystalline masses (see Fig. 5) which were found in some of the hæmorrhages, are points of pathological interest, which the limits of this communication do not allow me further to consider. I must, however, briefly refer to the hyaline degeneration which was such a remarkable feature both in this tumour and in a case which I saw with Dr Leslie, and which is reported on page 23.

Hyaline degeneration is met with in a great variety of different conditions. Recklinghausen, as the result of a long series of observations, concludes that the hyaline metamorphosis plays a considerable part in a great variety of different conditions, both normal and abnormal. It consists of a transformation (or infiltration) of the tissues, more especially the walls of the minute arteries, into a translucent, highly refractive material, which bears a close resemblance to the translucent material which is met with in cases of amyloid or waxy degeneration. It differs, however, in this important particular from the amyloid material, that it does not give the characteristic reactions with iodine and methy-violet.

Hyaline degeneration of the minute arteries has been observed in diphtheria, typhoid, scarlet fever, and other febrile affections; it is comparatively common in lymphatic glands; is frequently seen in the brain, more especially in the neighbourhood of caseous or hæmorrhagic foci. P. Meyer has shown that it plays an important part in the production of the minute aneurisms which are seen on the terminal branches of the pulmonary artery in cases of phthisis. The same observer has shown that true obliterative thrombi composed of hyaline material may frequently be observed in

infarctions of the spleen and kidney in cases of phthisis. In a case of chronic meningitis with caseous deposits in the pia mater, P. Meyer found at certain points, where the cerebral substance was in a state of hæmorrhagic softening, an infiltration of the walls of the vessels, and often a complete obliteration of their canals by the hyaline substance. He has also seen the afferent arterioles in the kidneys of scarlet fever completely obliterated by hyaline masses; and he records a very remarkable case, in which cerebral symptoms with high temperature developed in the course of an attack of acute rheumatism, and in which a most extensive hæmorrhagic softening of the greater part of the left occipital lobe was found after death, the cerebral lesion being apparently due to the plugging of the minute arteries with refractive hyaline masses.

The exact chemical composition of the hyaline material is unknown.

In some cases the hyaline deposits are, according to Recklinghausen, only products of the death agony, but in others, and amongst these there can be no doubt that the two cases of cerebral tumour to which I am now more especially referring must be included, the hyaline material had evidently been formed during life, and is of distinct pathological importance.

Various opinions have been expressed as to the source of this hyaline material. Almost all observers who have studied the subject seem to think that it is not simple fibrine. According to Recklinghausen, it is a derivative of cellular protoplasm. Most writers seem to think that the white corpuscles of the blood are the source from which it is usually derived, but P. Meyer and others admit that the endothelial cells of the bloodvessels, and in some cases, perhaps, all the elements of the tissues, may be transformed or fused, as it were, into hyaline material. Pitres believes that the hyaline material may be derived from the red blood corpuscles. P. Meyer does not think the evidence on this point conclusive; but I am disposed to think, for the reasons previously stated (see pages 40 and 41), that in the case related above (case of Miss A.) the large crystals, the irregular, transparent masses, and the hyaline material, were probably derivatives of the red rather than the white blood-cells.

Hyaline degeneration is seen where the conditions for satisfactory nutrition are interfered with; anything, whether a chemical or mechanical cause, or a dyscrasia, which interferes with the vitality of cell life, seems to predispose to its production. It is especially liable to occur in the neighbourhood of caseous foci, and in the cerebral arteries in cases of hæmorrhagic extravasation and red softening. Further, the two cases which we are now reporting show that an advanced degree of hyaline degeneration may be met with in cases of glio-sarcomatous tumours of the brain, and suggest the possibility of the hyaline change being a product, not merely

of the extravasated blood corpuscles, but possibly of the glial cells themselves.[1]

I regret that a microscopical examination of the blood was not made during life; and that the great viscera of the thorax and abdomen was not examined at the autopsy.

Dr M'Bride thought that although Dr Leslie laid stress on the absence of giddiness in his case, it was not so much a symptom to be expected in central as in lateral lesions of the cerebellum. Moreover, if he was right in his view that the diseases began some five or six years before death, it was possible to suppose that the other senses might, so to speak, become accustomed to the change, and act, if he might be allowed the term, vicariously. With reference to the difficulty of deglutition and of articulation apart from aphasia in Dr Bramwell's case, he thought that Krause of Berlin, in his experiments on dogs, found a cortical centre in the frontal region which so much influenced the bark as to be entitled to the name "centre for articulation." In the same region, too, Krause found a centre which governed deglutition.

Mr Cathcart remarked that the accurate observations of the effects of purgatives was of importance as throwing light on the question as to whether or not purgatives should be used in head injuries. Lately, some surgeons had objected to their use. Having himself seen benefit derived from them, he was interested to hear of their good effects in the cases described by Drs Bramwell and Leslie.

The President asked if the difficulty of articulation was due to a defective action of the lips, or of the tongue, or laryngeal movements, or a combination of these.

Dr Bramwell said the lips and tongue, not the larynx, were affected.

Dr Leslie said that he believed five years to have been the duration of his case, not quite so long as Dr M'Bride supposed. As to the suggestion that the giddiness might have been got over by habit, one would have expected to have seen some symptoms of the giddiness at first before it was got over in this way. He had recently a case under his treatment in which some of the symptoms closely resembled those of the case described by Drs Bramwell and Foulis. It was that of an old lady to whom he was called when she was in an aphasic condition, in which she stuck on the word "infective." She recovered from this attack in a few days, but during the next two years, till she died, had no less than twenty apoplectiform attacks, in which she appeared to be dying or dead. Constipation was a marked feature of her case also, but he found that extract of cascara had a wonderfully good effect.

[1] For further information as regards hyaline degeneration the reader is referred to Dr P. Meyer's paper, "De la formation et du role de l'hyaline dans les anevrisms et dans les vaisseaux" (*Archives de Physiologie normale et pathologique*, 1880, page 598 *et seq.*), to which I am indebted for many of the foregoing particulars.

Dr Bramwell was interested to hear of Krause's observations. He thought the absence of vertigo might perhaps be explained by supposing that the lesion was a "destroying" rather than a "discharging" one. The most marked case of cerebellar vertigo he had ever seen was examined last week. A secondary carcinomatous tumour occupied the same position in the right lobe of the cerebellum as the cyst in Dr Leslie's case.

Meeting III.—January 19, 1887.

Professor GRAINGER STEWART, *President, in the Chair.*

I. ELECTION OF NEW MEMBERS.

The following gentlemen were admitted Ordinary Members of the Society:—Robert S. Aitchison, M.B.; J. A. Armitage, M.B., C.M., Wolverhampton; J. Walton Hamp, L.F.P.S. Glasg., L.S.A. Lond., Wolverhampton; Wm. Hunter, M.D.; Sidney Rumboll, L.R.C.P. & S., Grangemouth; John Thomson, M.B., C.M.

II. EXHIBITION OF PATHOLOGICAL SPECIMENS.

1. *Dr Skene Keith* showed specimens of DISEASED OVARIES AND TUBES illustrative of his paper.

2. *Dr Bruce* showed A BRAIN taken from a child which had been under the care of Dr Carmichael in the Sick Children's Hospital suffering from what was diagnosed as simple meningitis. It seemed to be a perfectly typical case, and was shown as a contrast to cases of tubercular meningitis in the ward. Shortly after she was admitted she was seized with severe convulsions, and her life was saved by free bleeding from the arm. After that she made a steady recovery. She was dismissed sometime before Christmas as cured, and remained apparently perfectly well till a few days ago, when she was brought in suffering from general convulsions, and died a short time after admission. At the post-mortem an interesting condition of the membranes was discovered which fully confirmed the diagnosis. The surface of the brain was a little congested, but there was nothing remarkable in the membranes at the vertex. On exposing the base of the brain the arachnoid was found to be slightly thickened and milky over pons, sides of medulla, and cerebellum. There was a peculiar projection on the infundibulum, a little cyst with a clear colourless fluid, and on compressing the brain, the contents of this cyst could be increased. On looking at the under surface of the cerebellum and medulla, the fibrous thickening of the membranes had formed an adhesion between the two sides of the latter and amygdala,

which were also closely united. On opening into the brain the lateral ventricles were found considerably distended. The foramen of Munro was dilated to have a diameter of about half-an-inch. The ependyma over the lateral ventricles had a slightly granular appearance, and was slightly thickened along the superficial veins. The third ventricle was also dilated, as was the fourth. The pineal gland was normal, and the two veins of Galen not at all obstructed. It was probable that the increase of fluid was due to the blocking up of the lymphatic outlet, at the foramen of Magendie, caused by the gluing of the cerebellum to the sides of the medulla, and that no convulsion had occurred till the increase of fluid had become so great as to cause pressure on the floor of the fourth ventricle. The origin of the meningitis was supposed by Dr Carmichael to be rheumatic, and a slight confirmation of this was found in a fibrous thickening of one of the cusps of the mitral valve.

III. Original Communications.

1. REMOVAL OF THE UTERINE APPENDAGES FOR DISEASE, WITH TWENTY-THREE CASES.

By Skene Keith, M.B., C.M., F.R.C.S.E., Assistant-Surgeon for Ovarian Diseases, Royal Infirmary, Edinburgh.

For many years certain diseased conditions of the female pelvic organs have been known under the names of chronic cellulitis and chronic ovaritis, and have been treated by the application of blisters and nitrate of silver, with little permanent benefit to the patients; but, if we are to believe Dr Clifford Albutt, with golden benefits to the gynæcologists. Now, in addition to these old names and diseases, new pathological conditions have been recognised; or it would, perhaps, be more correct to say that a part of the body, hitherto thought little of, has been found to be sometimes the seat of grave and distressing diseases. Distension of the Fallopian tubes has been known for many years, but as a simple dropsy of these tubes seldom leads to disturbance, no importance was attached until lately to this part of woman. In late years it has become the fashion amongst a certain class of practitioners to lay far too great stress on the condition of the so-called uterine appendages, and to at once advise their removal when they are felt to be abnormal, or even indeed when they are not abnormal, but when, if the patient's statement is believed, they ought to be in a diseased condition.

The cause of this unfortunate state of affairs is twofold. In the first place, many of the gentlemen who remove ovaries either do not know what can be done by Nature, assisted by suitable local and general treatment, to clear up and dissipate pelvic troubles, or

because they are too impatient, and do not allow the *vis medicatrix naturæ* more than a few weeks to cure an ailment which may have been progressing slowly for several years. And, in the second place, the old dread of the peritoneum has been replaced by a confidence so great, that one abdominal surgeon has said that it is as safe to open the abdomen as to open one's pocket; and many have lauded the advice which has been given, to make a diagnosis after the abdomen has been opened.

Clinically, the various conditions for which removal of the uterine appendages may be required can be divided into—1st, Slight cystic enlargement of the ovaries, where the growths are enclosed in the folds of the broad ligaments, and which can scarcely be dignified by the name of ovarian tumours; 2nd, Salpingitis, hydro-, pyo-, and hæmato-salpinx, giving rise to symptoms, and where there is no other disease to account for the condition of the patient; 3rd, Where the uterine appendages have become compressed and bound down by adhesions; 4th, A combination of these three conditions along with a varicose state of the bloodvessels of the broad ligaments. The last is the most common. Under no circumstances is the operation to be performed for neuralgia, or unless disease is distinctly made out on examination.

A large part of the profession has not yet recognised these lately discovered diseases, and a still larger proportion will not entertain the idea of any but the old methods of treatment. This is not to be wondered at; for, like every new thing, this latest method of treatment by operation, or mutilation as it is often called, has been pushed too far, and ovaries have had to go which might quite well have been left alone. The symptoms of these diseases are also very diverse, and do not give any positive or definite information. You may have increased amount of the menstrual flow, or you may have a lessened amount; you may and generally do see emaciation, yet, on the other hand, the patient may be gaining weight; there may be leucorrhœa, or there may be no leucorrhœa; and even the only symptom which is never absent—pain—may give little information, for it may be worst before, during, or after menstruation, or even most severe between the periods. Its situation also varies, sometimes on the left side, sometimes on the right, or it may be worst in the back or across the front of the abdomen. The physical examination also is not easy, for there is always more or less tenderness; and it must be borne in mind that a distended Fallopian tube, or an ovary distended with pus, may be ruptured by rough handling. The only certain diagnosis is, however, to be made either and usually with the finger in the vagina—and I may remark that one finger is more likely to make it than two; or by the combined internal and external examination. It is not very difficult to feel a healthy Fallopian tube when examining a moderately thin patient; a healthy ovary, on the other hand, when in its natural position, is situated so close to the wall of the pelvis,

partially protected by the brim, that it can rarely be reached. An enlarged ovary can always be felt, as it is then altered in position.

When a distended Fallopian tube or diseased ovary, which is making life a burden to the woman, has been distinctly made out, and when the patient has been properly treated for chronic inflammatory disease in the pelvis, and is not any better for it, one of two things ought to be done: either leave the case alone, or remove the disease. The one exception to this rule, if exception it be, is when the patient has not been long married, and when the painful condition is believed to be kept up by too frequent intercourse. Here complete sexual rest for many months must be tried, along with general treatment, and followed, if a cure result, by increase of knowledge on things in general on the part of the husband.

Whether operative treatment is ever to be advised or not, must depend—1st, on the mortality following operation; 2nd, on the benefits to be derived from operation; and 3rd, on the drawbacks which follow operation. As the diseased condition of which we are treating seldom shortens life, a small mortality is absolutely essential. Sir James Paget has, I believe, said that a death-rate of 4 per cent. is too much for this operation; on the other hand, Dr Greig Smith, in a recent paper, has stated that the average operator will have a mortality of 8 per cent. If this latter statement be correct, the operation must be condemned, or the average operator must leave it alone. What the benefits which are to be derived from operative treatment are, we have at present not much reliable knowledge, beyond the meagre statement that the patients were cured, or, in other words, did not die, for these terms seem nowadays to be almost synonymous. To clear up this want of knowledge, at least in a small degree, has been my object in writing this paper, and I have given below the exact state of every case, twenty-three in number, I have operated on up to six months ago. The drawbacks to the operation are,—first, the convalescence is very slow; and, second, there is the idea that the patients are unsexed. The woman who has disorganized ovaries or occluded Fallopian tubes is already nearly as much unsexed as if these diseased parts had been removed. Nevertheless, it is absolutely essential that the patient be told that she will have no children. Husbands naturally wish to know how the operation will affect them. This most important question cannot be definitely answered, for I have been told by a woman, after her ovaries had been removed, that sexual relations, which had been impossible on account of pain, had been renewed, but that all desire had been lost. Two others have lost it, another remains as before, and in two cases of hysterectomy there has been little change, except that one has become more amorous, and this woman has not a morsel of uterus, ovaries, or Fallopian tubes. The Inquiry Com-

mittee appointed by the Liverpool Institute, *re* "Hospital for Women," makes this statement:—"A considerable proportion averred that they had suffered a distinct loss of sexual feeling, to such an extent as to cause serious domestic unhappiness in not a few instances." I know of another similar case.

Menstruation does not, I believe, recur when the whole of the ovarian tissue has been removed. When writing lately of the removal of the ovaries and tubes, a well-known London physician said, "The women had no disease, and they were cured." Surely on this question he has taken up an extreme position, forgetting that all extremes are errors.

It is difficult to give any idea of how often the operation is to be recommended. During the first year that my father and I began to operate on any but actually bed-ridden patients, we performed the operation twenty-four times. In the next six months not half-a-dozen women have required the operation. In fact, the supply had been almost exhausted by less than thirty operations. This fact makes one wonder at the numbers of diseased ovaries and tubes there appear to be in some parts of this country.

The operation itself may be very easy, or it may be very difficult. In a large majority of cases an incision long enough to admit two fingers into the abdominal cavity is sufficient, and no one who has not the manual dexterity to be able to remove the ovaries and Fallopian tubes through such an incision ought to attempt the operation. The fingers are passed down to the uterus, and along the broad ligament until the ovary, situated usually deep down in the pelvis, is reached. It is best to attack the one which is known to be least adherent first, so as to raise the whole pelvic floor, and thus bring the more difficult one more easily within reach. The ovary and tube are drawn outside the abdomen either by the fingers or by forceps; a double silk thread is passed through the broad ligament as far away from the ovary as possible, and is tied. The entire ovary is then cut away, care being taken to remove every morsel of it. Slight adhesions are easily broken down by the fingers; but when the appendages are embedded in a mass of adhesions, it is often most difficult to separate them, and, more especially, to know where to begin the separation. Bleeding is to be arrested by ligatures, by pressure, or by perchloride of iron, or occasionally a drainage-tube has to be left in, and the hæmorrhage will be found to gradually lessen, if no large vessel has been left without a ligature. The immediate after-history of the cases does not vary from that of a double ovariotomy. There is a discharge of blood from the uterus on the second, third, or fourth day, sometimes accompanied by considerable nervous disturbance. Hegar advises that the bowels be moved early, so as to prevent adhesion, and I can see no objection to this plan. There will be often a recurrence of the old pains at the date of the "periods," and it is as well not to allow the patient to sit up until after the first one be

past, and to warn her to be careful when the next ones become due.

Case I.—L. S., age 28, had suffered from dysmenorrhœa since the age of 14. Her present illness followed the birth of her only child twenty-eight months ago. Since then she has been unable to work, and has had four attacks of pelvic cellulitis, each of which kept her in bed for over a month. On one of these occasions I felt the uterus absolutely fixed by the surrounding swelling. She suffered from constant pain in both groins, bearing down, and pains in the back, all aggravated before and during menstruation. She has also menorrhagia and a profuse leucorrhœa, and is a martyr to facial neuralgia. She has not lost flesh. Operation on 10th August 1885. Dr Blair, of Shotts, wrote to say that he regretted that he was not able to be present at the operation, as he was anxious to see the last of the case, as it had been a constant trouble and anxiety to him for more than two years. Ovaries enlarged, and the tubes hard and slightly dilated; no adhesions. On 4th October the patient wrote to say that she was better than she had been for four years. *December* 1886.—Is in perfect health, and able to do anything, although she has had one severe hæmorrhage from the uterus, and three slight ones.

Case II.—Mrs N., aged 40, miscarried at the fifth month, nearly two years before operation, and as soon as she got out of bed felt severe pain in the left groin and across the abdomen. The pain has never left her, is aggravated before and during menstruation, and the general condition is much reduced. The tubes were easily felt to be enlarged and hard, and were removed on 25th August 1885. There were no adhesions. Slight cellulitis followed the operation; but when the woman went home at the end of five weeks she felt "quite different." *December* 1886.—She has no pain, and is well.

Case III.—Mrs L., aged 37, had suffered for nine years from pain in the back, in both groins, and from cramps and pains in the thighs, worst for a week before menstruation. She had had nearly a dozen doctors to see her at one time or another, and had spent most of the last two years in bed. On the 29th August 1885 the ovaries were removed with difficulty on account of pelvic adhesions; they were large, and the tubes were remarkably hard. Six weeks after the operation she said that her sight and memory were improving, that she was entirely free from pain, and that she felt her mind stronger. *November* 1886.—Perfectly well.

Case IV.—Mrs G., age 34, had been slowly getting weaker for five years since the birth of her fifth child, and now says that, except that she is alive, she is of no use. She has pelvic pains, but these

are bearable. Menstruation is profuse. On the 2nd September 1885 I removed the ovaries; the right ovary and tube were much enlarged, and had been easily felt before operation. They were adherent to the omentum and in the pelvis; the left ovary and tube were removed, though they were healthy. After going home to Glasgow convalescence was retarded by an attack of acute nephritis. In the beginning of May 1886 she felt well, but not very strong. On 13th July she spent a long day in the Exhibition, and, as she is still as well as she was then, I think she may safely be said to have made a complete recovery.

Case V.—Miss L. B., age 31, looking wan and thin, was unable to say how her illness began, but had suffered from pains in both groins and in the back, and from leucorrhœa, for fully seven years. A large swelling was to be felt behind the uterus on the left side, and a smaller one on the right. On 3rd September 1885, the right ovary and tube were found to be enlarged and adherent; the left ovary had been transformed into a thick walled cyst almost as large as a billiard ball. The operation was followed by slight phlegmasia dolens, causing stiffness of the leg for some months. In November 1886 she was in magnificent health.

Case VI.—Mrs C., age 35, dated her illness to the birth of her last child six years ago. She suffered from constant pain in both groins, which was worst for two days before and for the first two days of the menstrual flow, and from great and increasing debility. She was much emaciated, and her friends told me that they noticed her becoming thinner almost day by day. Greatly distended Fallopian tubes were to be felt. Operation on 12th September 1885. The right tube was filled with serum, and was as large as a distended small intestine; the left ovary and tube were matted together into a mass nearly as large as a hen's egg. There were pelvic and omental adhesions. In December 1886 the woman was quite well.

Case VII.—Mrs G., age 41, suffered from pain in the left groin for fifteen years. For the last two years she has been unable to work, and has been as long as five months in bed at a time. There is menorrhagia, and the pain is worst at the end of the flow. The uterus was large and retroverted, and the left ovary was enlarged. 30*th September* 1885.—Appendages removed, pelvic adhesions; left ovary had become transformed into a single cyst; the right was large, and the tubes were healthy. She was very weak, and on the second day the temperature was 103°, pulse 140, and respirations only 10, and after several ups and downs she got home in two months. In January 1886 she wrote to say that she was well; in March she was worse; then in April she was better. Now, fifteen months after the operation, Mrs G. is little, though certainly a little better than she was before.

Case VIII.—Mrs Y., age 35, had suffered from pain in the left ovarian region for twelve years, since the birth of her second child. At first the pain was not constant, but for years it has never left her. There is also pain in the right side, and for the last two years the worst pain has been in the back. For ten years the patient was almost constantly under medical treatment, and, after seeing a larger number of doctors than she can even remember the names of, and having been an in-patient in the Royal Infirmary on two occasions, she came to the conclusion that she was incurable. After an interval of two years, and on account of frequent attacks of vomiting being added to her troubles, she thought that she would make another attempt to gain relief; and now, after thirteen years of suffering, I think she is to be well. There was no difficulty in feeling the enlarged tubes through the abdominal wall, and from the vagina the exquisitely tender masses were easily made out. The operation was performed on the 6th October 1885, and was a most difficult one. The left tube and ovary were the more difficult to remove, and, unfortunately, I was unable to cut away the whole of the organ on that side—a morsel being left beyond the ligatures, which I did not dare to remove for fear of slipping of the loops of thread. The patient drove home, a distance of eight miles, at the end of three weeks; she was very happy, and had no pain. Two days after going home she had to go to bed with severe pain in the left side, and on the thirtieth day after the operation there was a profuse red discharge from the uterus. I examined, and found a hard mass on the left side extending up to the anterior superior iliac spine, evidently a hæmatocele. By the end of January she was feeling much better, and in a fortnight her husband came up to say that she was splendidly well, and that there was no pain. She kept fairly well till April, though menstruation recurred usually every three weeks. At this time vomiting began to be frequent, everything was bitter, and the tongue was large and indented by the teeth. In the beginning of August she was steadily improving; the periods were regular, there was less discharge, and little pain. This was the last good news, and in October I had to take her back into the hospital; the pain was as bad as it had been before the operation, and the vomiting and sickness were very much worse—indeed, she was sick after almost every meal. There was still a swelling on the left side of the uterus, and I suggested that an attempt should be made to remove the small piece of ovary which was known to be there. The patient and her husband at once consented, and after waiting nearly three weeks for a day when she was not sick, I cut down again, reached the mass on the left side, and after a great deal of difficulty was able to remove a tumour the size of a small hen's egg. This mass was universally adherent, and burst, part of the contents being old blood-clot. The case was drained, and there is only one faint cicatrix, barely two inches in length, to be seen on the abdomen. No

sickness, pain, nor uterine discharge followed the operation, and the woman was kept on her back for five weeks. Four more weeks have now passed, and there has been no menstruation; "flushings" are troublesome, but otherwise the patient is very well, and takes her food better than she has done for years.

Case IX.—Miss A. B., age 36, when 11 years of age fell off a hay-cart. This fall brought on her first menstrual discharge. She says that she screamed for five days with pain in the back. There was dysmenorrhœa for eight years; she was better for two; then had a baby, and for fifteen years has suffered from dysmenorrhœa, the pain coming on the third day before menstruation. On the day before the flow appeared the pain amounted to agony, and she often rolled on the floor. With the appearance of the flow the pain lessened, but she then suffered from frightful headaches, lasting a week. She constantly lived in dread of what each month was to bring. *7th October* 1885.—Right ovary cystic; left hard and fibrous; tubes dilated; pelvic adhesions. A few days ago the patient said that she was quite well.

Case X.—Mrs M., age 29, complained of constant pain in the left side and back, of menorrhagia, and of a steady decrease in health and flesh. She dated her troubles to the birth of her last child seven years ago. Operation on 28th October 1885; no adhesions; ovaries large, tubes healthy. On the anniversary of her operation she called to say that she was quite well.

Case XI.—Mrs G., age 26, had suffered from pain across the lower part of the abdomen for six years since the birth of her first child. The pain was at its worst for two days before menstruation. A second child was born four and a half years after the first, and since then she has been much worse, and has been unable to do her housework. She is very thin. The ovaries were felt to be enlarged, and the left was diagnosed to be adherent. This was found to be the case on 26th December 1885. Is now in the most perfect health.

Case XII.—Mrs G., age 34, dates the commencement of her troubles to the birth of her second child eleven years ago. It is now four and a half years since her youngest was born, and for this length of time she has never known what it was to be free from pain, which is worst before and between the periods. She is wretchedly thin, and has had nine months of treatment, which she describes as "being burnt inside," and was none the better for it. Operation on 5th January 1886. There were no adhesions. After leaving hospital her convalescence was not hastened by being turned out of the house one night by her drunken brute of a

husband. She is now working all day long charing; she has no pain, and is well.

Case XIII.—Mrs H., age 34, fell off a chair ten days after her last child was born five years ago. She was lifted into bed, and at the end of six weeks was removed to the Infirmary. While there, an abscess was opened in the left groin, and she was also "burnt inside." The pain, which she has now had for five years, is situated principally in the left side and back, and is worst before the periods. There is profuse leucorrhœa, and the menstrual flow has lessened in quantity. The operation on 25th January 1886 was complicated by adherent intestine preventing the passage of the fingers into the pelvis; the right ovary was cystic, the left small and cirrhotic, and both were adherent in the pelvis. Five weeks afterwards she looked fat and well, and although complaining of some of the old pain in the left side, had been able to paper one of her rooms unaided. Last week she wrote that she was happy to say that she had not the constant pain she had before, but still has a great deal at certain times.

Case XIV.—J. D., age 27, "racked" herself seven years ago while nursing her mother. The pain was first worst in her left side; now the right is the worst. The pain commences to increase eight days before menstruation, and lasts through it. This patient came to the hospital several times, and at last, though the case was not looked on as a favourable one, the operation was performed on the 5th February 1886. The left ovary was cystic, had split up the broad ligament, and was removed with great difficulty. The right ovary had become transformed into a cyst containing ʒvi. of clear yellow fluid, and was entirely enclosed in the broad ligament. The enucleation was very difficult, both on account of the thinness of the cyst wall and from the close adhesion, and it was not possible to say whether the whole organ had been removed or not. This was the only case of the series where I had to make an incision larger than would admit two fingers. The operation has been a complete failure, and the girl now menstruates oftener than she used to do.

Case XV.—Mrs T., age 28, never pregnant, for six years has suffered from pain in both groins, in the back, from leucorrhœa, and most especially from dyspareunia—the latter, with loss of strength and emaciation, being the most prominent symptoms. Three years ago she had gonorrhœa. The operation was on the 12th February, and it was expected that pus would be found in the right tube. Instead of this the ovary was cystic, and contained ʒv. of fluid. There were adhesions, and the broad ligament was opened up. The left ovary was also adherent and enlarged, and the tubes were not healthy. In the beginning of June the patient

returned from a visit to Ireland, and was then very well. In September she complained of pain in the left side; but had been travelling too much, and there was nothing to be felt. In the beginning of November there was a slight bloody discharge from the uterus; and on December 17th she again came into town, complaining of pain in the left side. A hæmatocele extending as high as the anterior iliac spine was then felt, and with rest the pain is abating.

Case XVI.—Mrs J., age 37, suffered from constant bearing down for twelve years since her first child was born; with the birth of each child the pains have become worse, and for four years she has had, in addition, pain in both groins. She had been an in-patient in the Glasgow Royal, Western, and Edinburgh Royal Infirmary, and had been discharged from each, no better. On examination, the uterus was found to be retroverted, due probably to a small fibroid on the fundus; the right ovary was enlarged and adherent, and the left tube was thought to be dilated, though this turned out to be the ovary elongated and cirrhotic. On the 6th March the left ovary and tube were removed as usual, and the broad ligament below the right was secured in a clamp, so as to prevent recurrence of the retroversion. In the beginning of November the patient started with her children to join her husband in India. This she had desired to do for some years, but had never felt well enough for the journey.

Case XVII.—Mrs D., age 34, suffered for eight years from pain in the back and left groin, and from leucorrhœa, and lately has been losing flesh. The uterus was large, cervix torn, and the right ovary was tender, adherent, and enlarged. As sewing up the cervix would have improved her general condition, and probably cured the pain in the back without relieving the ovarian pain, I removed the ovaries and fixed up the uterus, as in the preceding case, on 26th March. The tubes were distended by fluid. Local and general treatment had previously given temporary relief. On 13th August patient sailed for America with five children.

Case XVIII.—Mrs H., age 48, suffered from constant pain in the right side and from prolapse for eleven years. She feels life a burden, and has been unable to wear a pessary on account of the pain in the right side. As the right ovary and tube were felt to be enlarged and adherent, a similar operation to the two previous ones was performed on 23rd April 1886, and by August the patient was fit for anything, and continues well.

Case XIX.—Mrs R., age 22, was delivered of her first and only

child five years ago, and has not been well since. She has constant pains in both groins and across the front of the abdomen, worst usually while menstruating. Dsypareunia is also a prominent symptom. Her husband, a military gentleman, gave her syphilis shortly after marriage. She had been seen by two specialists on removal of the appendages, who were said to have diagnosed syphilitic disease of those parts, whatever that may mean. The appendages could be easily felt, and were exquisitely tender. They were removed on 27th April 1886. In September she was well and strong, but still had some pain in the left side, which had, however, disappeared in December.

Case XX.—Miss C., age 28, has been ill for seven years with pain in the back and sides, and has seldom been out of the doctor's hands. Various kinds of pessaries have been tried, but could never be kept in, as they always caused pain at one particular spot behind the uterus. The uterus was large and retroverted, and behind it the right ovary, painful and tender, could easily be felt; the left ovary was also large, but appeared to be not adherent. On 27th May the condition was found to be as described, except that the left ovary was adherent. The uterus was fixed in the wound in the manner already described. When she went home, in seven weeks, she felt like a different being, and could hardly believe that she was free from pain. She is now well.

Case XXI.—Mrs M., age 26, had constant pain in the back and sides for six years, increased menstruation, and had become much emaciated. As, in addition to retroversion of the uterus, there was enlargement of the ovaries and tubes, those organs were removed, and the uterus was fixed up with a clamp. In this case the veins in the broad ligament were enormous. In December 1886 she was very well.

Case XXII.—Mrs C., age 34, for some years had suffered from menorrhagia, which was steadily becoming worse. In addition to this, there was constant pain in the ovarian regions. She said that she could have got along with one, but that the hæmorrhage and the pain were becoming too much for her. As general treatment, followed by curetting, give but very temporary relief, I removed slightly enlarged ovaries and tubes on 6th July 1886. The only particular point of interest about the operation is, that, owing to the thin and lax condition of the abdominal wall I was able, after separating pelvic adhesions, to draw the ovaries so far up as to sever their connexions by the cautery. This patient is now quite well.

Case XXIII.—Mrs B., age 37; it was found to be impossible to

liberate the appendages from adhesions, and the operation had to be abandoned.

Looking at the results of these twenty-three operations, and it is probable that those, in most of the cases, are the ultimate results, we find that eighteen, or seventy-eight per cent., have been cured, and that two, or nearly nine per cent., have received no benefit from the operation—one of these being what is now called an exploratory incision, that is, a failure or mistake in diagnosis. Of the three remaining cases, one has a hæmatocele, which did not form until seven or eight months after the operation, and there is no reason to suppose that the patient will not be quite well when this has disappeared. The second is improved, and the other is the most disappointing, though I believe that in time she will become quite well, and that, if she had been in better circumstances, her long illness would now be a thing of the past. Five were non-adherent, and twenty-one had received treatment previously for periods of time varying from nine months to twelve years—eight of those having been hospital in-patients.

A pregnancy or abortion was stated by sixteen to have been the cause of the disease, two ascribed it to an injury, and the others were unable to say more than that their illness had gradually come on. Four had never been pregnant, but one of those was married.

Menstruation has not appeared in any of the complete cases, though in Case I. there have been on four occasions a hæmorrhagic discharge from the uterus; and in Case XV. there was a slight red flow for a day or two at the time of the internal hæmorrhage. Before operation, the menstrual flow was increased in nine, diminished in four, and in ten there was no change.

Since the beginning of July of last year I have performed this operation four times—two of these having been previously hospital in-patients, one treated by an hospital obstetric physician, and the fourth had been sent from Orkney; and as I have not been so unfortunate as to lose a case, my operations have been justifiable on the first question, that of the mortality; the relief of pain and the improvement in the general condition have also justified most of the operations, and as yet I do not know of any drawbacks which have followed. The great difficulty is to know where to draw the line between operation and no operation; but I am certain of this, that, as long as the passing of a uterine sound is considered to be a part of an ordinary examination, so long will there be inflamed ovaries and Fallopian tubes to be removed.

A study of those and other cases has also shown me that some women at least get far too much local treatment, more especially of that kind which is commonly described by patients as being "burnt inside."

No.	Date.	Residence.	Age.	Adhesions.	Result.
	1885.				
1	August	Hospital	28	None	Cured.
2	,,	,,	41	None	,,
3	,,	,,	37	Pelvic	,,
4	September	,,	34	Pelvic and omental	,,
5	,,	Private	31	Pelvic	,,
6	,,	Hospital	35	Pelvic and omental	,,
7	,,	,,	41	Pelvic	Little better.
8	October	,,	35	Pelvic. Second operation, 19th Nov. 1886	Cured.
9	,,	Private	36	Pelvic	,,
10	,,	Hospital	29	None	,,
11	December 1886.	,,	26	Pelvic	,,
12	January	,,	34	Pelvic	,,
13	,,	,,	34	Pelvic and omental	Improved.
14	February	,,	27	Pelvic. Broad ligaments opened up	No better.
15	,,	,,	28	Pelvic. Broad ligaments opened up	Hæmatocele.
16	March	,,	37	Pelvic	Cured.
17	,,	,,	34	Pelvic	,,
18	April	,,	48	Pelvic	,,
19	,,	,,	22	Pelvic	,,
20	May	,,	28	Pelvic	,,
21	June	,,	26	Pelvic	,,
22	July	,,	34	Pelvic	,,
23	May	,,	38	Deeply imbedded. Not removed	No better.

Dr Brewis remarked that a great deal had been said lately about the justifiability of this operation. They might, however, agree that in properly selected cases it was justifiable. The difficulty of selection arose partly from the present methods of examination not being sufficient to enable them to detect the exact condition of the uterine appendages. Bimanually they could make out enlargements of ovaries and tubes, but an ovary might be diseased and not enlarged. The symptoms might then help. Putting aside pain, which was very valuable, that of sterility was important. When a woman who had had children was sterile for some years, the uterus or appendages might be considered to be at fault. Bimanually they could thoroughly examine the uterus. If it was right they might conclude that the uterine appendages were at fault. If the sterility existed along with other symptoms which rendered life miserable or in danger, they were, he thought, justified in recommending the operation.

Mr Cathcart asked Mr Keith what his views were as to the pathology of the condition. Previously he thought many of these cases were subsequent to gonorrhœal inflammation. He was

interested to notice that the majority of Mr Keith's cases followed abortion or childbirth. He should like to ask if there was evidence of any septic change having occurred, which had become chronic.

Mr Annandale said he had seen lately an explanation of the parotitis following ovariotomy, which was to the effect that the removal of the ovary caused some interference with the salivary secretion, and the ducts in such a condition permitted the entrance of germs to the glands, and so caused the inflammation. He wished to ask Mr Keith if he had met with any case in which this might be a likely explanation of this condition.

Dr James Ritchie asked if Mr Keith had any information as to the proportion of his cases in which the sound was passed. Judging from his experience of the practice in the gynæcological wards of the Infirmary some years ago, the passing of the uterine sound was a frequent operation, and if they were to take it as true, that this severe pathological condition calling for the removal of the uterine appendages was so rare, that Mr Keith and his father had, in a short time, practically exhausted the cases in this district, he could hardly believe that the passing of the uterine sound could be a common cause. He was of opinion that the most common cause was gonorrhœa, or a septic inflammation. He was surprised to note that in so large a proportion of the cases there was neither metrorrhagia nor menorrhagia, which were described as symptoms.

Mr Skene Keith, in reply, said that it was extremely difficult, if not indeed impossible, to say when a woman was sterile. A patient had come under his care lately with what appeared to be undoubtedly distended Fallopian tubes. Her illness had followed a miscarriage five years before. With rest and general treatment the patient improved and became pregnant for the second time, but aborted at the third month, and now the tubes were felt to be quite small and soft, and her old pains have gone. He did not think that gonorrhœa, at least in this part of the country, had much to do with the disease. In one case only was there a history of gonorrhœa, and in that case there was no pus, nor was there any in the case of the patient with syphilis. The sound had been passed in twenty-one of the twenty-three cases. He thought the condition was inflammatory, and that laceration of the cervix had often more to do with it than anything else. He was struck with the little change in the menstruation to which Dr Ritchie had referred. It had at first been given out that there was always increased menstruation, but the symptoms had then not been fully worked out, and in some cases there had doubtless been increase, which may have led to the belief that it was always present. He had seen suppuration of the parotid after ovariotomy. He went into the question pretty fully, and believed that it was as common after other operations.

Mr Annandale said it was inflammation, not suppuration, he referred to.

Dr Ritchie said it was held that inflammatory affections following confinement might be traced to a gleet in the husband which had not caused mischief till that time.

Mr Keith thought that in that case the trouble would have shown itself immediately after labour, but all his patients made a good recovery from the labour, and did well till they got up. He believed that a torn cervix had more to do with it than the gleet. This was shown in one case which had been sent in for operation. The torn cervix was sown up, and the operation was followed by softening of the tubes, which had been enlarged and hard, and the patient had been completely cured.

2. NOTE ON PRELIMINARY TRACHEOTOMY AS AN AID TO CERTAIN OPERATIONS.

By Thomas Annandale, F.R.C.S. Ed., Regius Professor of Clinical Surgery, University of Edinburgh.

In this note I desire to add my testimony to the value of performing preliminary tracheotomy in certain operations involving the mouth, throat, or jaws, and also to describe a simple appliance which I have found from experience to be most efficient in carrying out the principles of this proceeding.

All practical surgeons acknowledge the importance of this aid in suitable cases, and my attention was first seriously directed to the subject in connexion with several cases of accidents occurring during the progress of an operation from blood passing into and obstructing the air-passages. The patients in these cases were under the influence of an anæsthetic, and their lives were only saved by prompt tracheotomy, and the removal of the obstructing blood and clots.

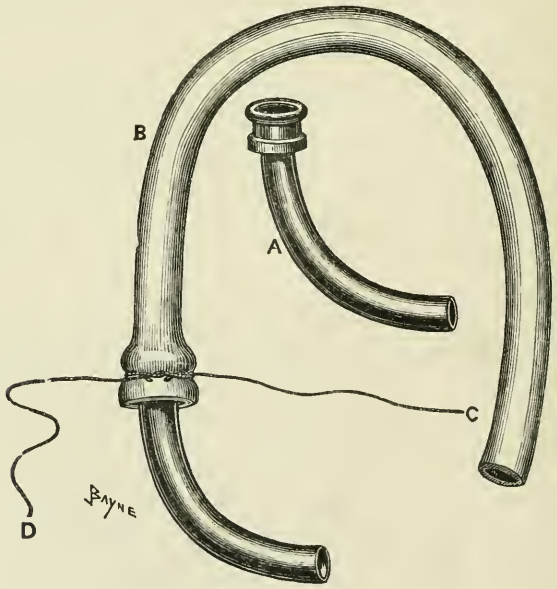
The occurrence of these cases and a knowledge of others leads me to urge that preliminary tracheotomy should be practised perhaps more frequently; and my own experience of this aid to operating in cases which are likely to be attended by hæmorrhage into the air-passages has given me confidence in practising it, and also in advising its practice by others.

The two important principles of preliminary tracheotomy in the class of cases referred to may be briefly stated as,—(1.) The prevention of blood passing into and obstructing the air-passages; (2.) The security of free and proper respiration, together with the safe and convenient administration of an anæsthetic. In addition, I think it is as well that much blood should not pass down into the stomach, as it may cause vomiting, and so interfere with the operation.

In the *New York Med. Journal* for March 24th, 1883, there is an excellent practical paper upon the subject by Dr Charles M'Burney, and also a note of a discussion upon his paper. Dr M'Burney refers to the objects of the operation, and agrees that it can be carried out efficiently by an instrument more simple than that of Trendelenburg. It is generally admitted that the instrument of Trendelenburg, and also the modifications of it, although most ingenious, are not very satisfactory in practice, as they may readily get out of order, and be useless to carry out efficiently what is required of them. Accordingly, those surgeons who have practised preliminary tracheotomy in recent times have endeavoured to employ some more simple apparatus, and I will now describe the one which I have used with perfect success, and to which, therefore, I direct the attention of my professional colleagues.

It has been suggested that the preliminary tracheotomy should be performed some days or weeks before the major operation, but most surgeons are, I think, agreed that this is quite unnecessary, and that the best time to perform it is immediately before the operation which it is desired to aid.

I prefer to open the trachea at its upper part above the isthmus of the thyroid; but if any condition prevents this, I would then perform laryngotomy.



My apparatus, figured in the woodcut, consists of a vulcanite tracheotomy tube (A), which has a broad ring attached to, but

moving round its neck, and of a piece of ordinary india-rubber tube about eighteen inches long, and having a diameter of half an inch. One end of the india-rubber tube (B) is passed over the ring at the neck of the tracheotomy tube, and secured to it by a piece of strong silk, the ends (D and C) of which are left long. The trachea or larynx having been opened, the tracheal tube is introduced as far as its neck, and then each end of the silk is, by means of a needle, brought through the corresponding edge of the wound in the skin, and when these are tied together they not only retain the tube in the trachea, but also close the external wound, and prevent any blood passing into the trachea in this way. The movable ring round the neck of the tracheal tube has the advantage that it allows of some corresponding movement of the tubing connected to it, so that the latter is much less liable to be twisted or bent, and its canal to become obstructed.

To administer an anæsthetic, the free end of the tubing is introduced into a glass or tumbler, in the bottom of which a little cotton wadding is placed, and upon this cotton wadding chloroform or ether is dropped from time to time as may be required to keep up the anæsthesia. The tube and glass are held by an assistant at one or other side in the position which least interferes with the manipulations of the operator.

Immediately previous to the major operation, and after the tracheotomy has been performed, the throat is thoroughly stuffed by means of a piece of sponge or some cotton wadding, which should be *covered by oil-silk or other thin waterproof material*, in order to diminish the chance of any blood oozing through the pad. It is well to attach to the pad a piece of strong silk or thread for the purpose of withdrawing it more readily.

Should the bleeding have ceased and there be no risk of swelling or œdema in the neighbourhood of the glottis, the tracheotomy tube may be removed immediately after the completion of the operation, but if these favourable conditions are not present it is safer to leave in the tracheal tube, detaching the tubing from it, for twelve or twenty-four hours, or for a longer time, according to the symptoms. I would like further to add that on three occasions lately I have, in order to facilitate the removal of large and vascular tumours involving the mouth and throat, not only performed preliminary tracheotomy, but also preliminary ligature of the corresponding common carotid artery. All these cases were successful, and I attribute much of their success to these preliminary operations.

Dr Duncan said it was necessary to open the windpipe on certain occasions where the entrance of hæmorrhage into the air passages during the performance of operations was to be feared. In most cases position might do a great deal to obviate these dangers. He himself preferred the side position to allow the

blood to run out of the mouth. Mr Annandale was in the habit of operating with the head thrown back over the table, which prevented the passage of blood into the trachea, but he thought it rather apt to increase the blood flow. But there were cases undoubtedly, though not so many as Trendelenburg would make out, in which preliminary tracheotomy was beneficial. He had the other day an epithelioma of the tonsil, a disease in which there was a tendency to considerable hæmorrhage from vessels not easily got at, especially in a cavity where one could not be absolutely sure of cutting very wide at all parts of the growth. In that case he performed a preliminary laryngotomy, the opening of the larynx being an easier and quicker operation than the high tracheotomy. It was not a matter of great importance whether the one or the other was done. As Mr Annandale had said, the proper period for its performance was immediately before the major operation. There could be no advantage from doing it sooner, and disadvantages, such as bronchitis and pneumonia, might arise. An important point, and Mr Annandale had laid down the laws for meeting it, was when the tube should be removed. Where there was risk of œdema glottidis it had better be kept in during the next day or two days, but if the operation were not at the root of the tongue or tonsil, or back part of the pharynx, the tube should be removed immediately after the operation, as soon as the risk of blood getting into the air passages had ceased. The prevention of the entrance of blood into the air passages was best done by the sponge. A small sponge was all that was required. The only advantage of the large sponge was that it could be easily got out, but this could be done as well by means of a string attached to the small sponge. The disadvantage of the sponge was said to be that it might interfere with the operation, but even in the case of removal of the tonsil he had not found it so.

Dr Scott Lang asked if Mr Annandale had ever used the Trendelenburg tampon. It seemed to him that it would not prevent the lodgment of clots in the box of the larynx. He thought this was a disadvantage which the methods recommended by Mr Annandale and Dr Duncan got rid of. He wished to know how and when the clots were to be got out of the larynx, supposing an operation to have been completed, using the Trendelenburg apparatus.

Mr Cathcart asked if Mr Annandale's experience corroborated what he observed at the only operation at which he had seen the tampon used. After the introduction of the tube and inflation of the balloon, there was much more coughing than occurred after the introduction of the ordinary trachea-tube. This prevented the operation going on for an unusually long time, and was attributed to the irritation caused by the pressure of the balloon on the mucous membrane of the trachea.

Mr Annandale said he was glad to think the paper was received by surgeons as laying down a proper practice. It was a fact that,

within the last few years, he had begun to place the head over the end of the table in the operation for cleft palate. He did not think there was much difference between the two positions, but in the one case of cleft palate, in which he had such trouble from bleeding into the air-passages, as was described, the head was on the level of the table. He did not care for the sponge alone. It was too porous; but he preferred it covered with india-rubber or waterproof material. He had never used the Trendelenburg tampon. It was in his museum as a specimen of the kind of instrument, which, although ingenious, could not be used satisfactorily in practice.

Meeting IV.—February 2, 1887.

Professor GRAINGER STEWART, *President, in the Chair.*

I. DEATH OF THE OLDEST MEMBER.

The President said that he thought it desirable, before entering upon the business of the evening, to allude to the fact that since the last meeting a much-esteemed member of the Society, Mr William Brown, had died. By the death of this gentleman the Society had lost the last of its original members. Early in its history Mr Brown had held the office of Secretary, and subsequently that of Vice-President. It was unnecessary to speak at length of his efficiency as a professional man, his general culture, or his long career of Christian activity, but the disappearance of the last of their original members was worthy of special notice.

II. ELECTION OF ORDINARY MEMBERS.

The following gentlemen were admitted Ordinary Members of the Society:—George Franklin Shiels, M.B., C.M.; T. Brown Darling, M.B., C.M.

III. EXHIBITION OF PATIENTS.

1. Cases of EMPYEMA were shown by *Drs Affleck, Cotterill, Russell, Duncan,* and *Caird.*

Dr Affleck showed a YOUNG WOMAN upon whom the operation of RESECTION OF RIBS had been performed for the cure of empyema, with entire success. The patient was admitted to the Royal Infirmary in June last, suffering from a well-marked empyema of the left side, which was discharging by four openings in the thoracic wall. It appeared that she had been ill for about eight

months—first with pleurisy with effusion, which became complicated subsequently at short intervals by pneumonia, scarlet fever, and erysipelas. Empyema was the result, and an opening had been made to evacuate the pus, after which other three openings took place in the chest wall, and each of them discharged freely. There was also a fistulous opening into a bronchus, as large quantities of pus were daily expectorated. After continuing under treatment in Dr Affleck's ward for some weeks, the patient was removed in the beginning of August to Prof. Annandale's ward, where Mr Cotterill performed the operation of removing portions (each about 4½ inches in length) of five ribs. The patient rapidly improved and ceased to expectorate, while only a little pus came through the drainage tube, and this gradually diminished, till now it had entirely ceased and the wound had healed up. The chest wall had fallen in considerably, but yet good breath sounds were audible over the upper third or more of the lung. The patient had gained about two stones in weight, and was about to return to her occupation as a domestic servant. The case appeared in every way a suitable one for surgical treatment, there being no evidence of phthisis, nor of kidney or other complication.

Dr Cotterill showed the PORTIONS OF RIBS removed.

Dr Russell showed a CASE, the details of which appear in his paper.

Dr Duncan showed TWO CASES OF EMPYEMA at present under his care in the Infirmary. One was the third successful case after resection of portions of eight ribs. He had before that resected portions of three ribs, which was not entirely satisfactory. He had five times resected portions of eight ribs, three of the cases being quite satisfactory, one nearly so, and the other died of pyæmia after having gone home, where a drainage-tube was allowed to slip into her thorax. In the case shown the resection was followed by absolute healing, but about a fortnight or three weeks afterwards an abscess formed, and required the removal of a strip of bone from the chest wall, a very difficult thing to do, inasmuch as the front of the chest was one mass of bone. It was now practically healed. The lower ribs which were not resected bulged out, and the spine showed lateral curvature. There was breathing in the upper part of the lung. The second case was one of simple empyema without resection of ribs. A complete cure had been brought about in seven weeks by drainage. A rib was resected because the drainage was thus freer, and the severe pain which sometimes resulted from the pressure of the tube in the intercostal space was saved. The lung had expanded, and a point of interest was that the wound throughout the whole course of treatment remained aseptic without the spray.

Mr Caird showed a CASE OF RESECTION FOR EMPYEMA in a child. In summer 1885 the pleural cavity was drained with antiseptic precautions, and duly healed. The patient was in poor circum-

stances and bad health, and reappeared with the sinus opened up. A metal tube was introduced, and washing out practised without much improvement. Accordingly, a year ago, resection of portions of the fourth, fifth, sixth, and seventh ribs was performed, a counter opening made, and a good result ensued.

2. *Dr P. H. Maclaren* showed a MAN, aged 19, presenting 132 cutaneous tumours all over his body, except on the palms of the hands and the soles of the feet. The case looked like one of fibroma molluscum, but when some of the tumours were removed their contents were seen to be sebaceous. No openings could be detected anywhere in connexion with the tumours, and pressure could not expel any fluid or solid material, thus differentiating the case from so-called molluscum contagiosum. These cases were very rare. Hebra had seen one, Rayer also had seen a case, and Dr Allan Jamieson had described one in *Ed. Med. Jour.* 1873, in which there were 250 tumours scattered over the body.

3. *Dr Cotterill* showed a CASE OF COMPOUND COMMINUTED FRACTURE OF UPPER AND LOWER JAWS. The patient, a young man, had fallen from a roof, and the blunt spike of a railing had entered outside the left eye, and had torn the hard and soft tissues obliquely across the whole breadth of the face. The external angular process of the frontal was broken off, the left malar bone was smashed into several pieces, the superior maxillæ on both sides were comminuted and completely divided in a horizontal direction backwards to their posterior limits, so that the alveolar edge and palatal processes, with the palate bones, fell away from the upper fragments of the maxillary bones. The hard palate was also split into two halves in the antero-posterior direction, and the teeth of the upper jaw lay over the chin. The lower jaw was comminuted near the angle on the right side. Every fragment of bone which was attached to periosteum was left, and these fragments were fastened together with some twenty silver sutures. The lower jaw was drilled and brought together by wire, and loops of wire put round various of the teeth in upper and lower jaws to keep the bones in position. The man had made an excellent recovery with comparatively little disfigurement, while all the fragments of bone wired together had survived with the exception of two or three very small pieces.

IV. EXHIBITION OF PATHOLOGICAL SPECIMENS.

1. *Dr Littlejohn* showed—(1.) THREE SPECIMENS OF AORTIC DISEASE, in all of which sudden death had occurred. The various stages of atheroma were well marked. (2.) A FRACTURE OF THE SHAFT OF THE HUMERUS close to the joint, and attended with much displacement. The specimen had been taken from the body of a gentleman,

much decomposed, found in a house where it had lain for at least six weeks. (3.) THE STOMACH, INTESTINES, GULLET, AND WINDPIPE of a man who had swallowed a quantity of Bow's liniment. There was marked evidence of intense irritation.

2. *Dr Woodhead* showed—(*a.*) the LUNG, PLEURA, and CHEST-WALL from a case of EMPYEMA. The empyematous cavity extended up as far as the apex of the lung and down to the angle between the diaphragm and the ribs. It had been drained at the level of the eighth rib. (*b.*) SPECIMENS of TUBERCULAR PLEURISY.

3. *Dr H. A. Thomson* showed—(1.) A STUMP from a healthy man aged 43, amputated at the middle third of the leg four years ago for railway injury. It had healed well, except a small area in centre of face of stump, which never cicatrized. As he suffered much pain from ulcer, reamputation was performed 3 inches higher up. On dissection the nerves were found at the point of section to have formed bulbous enlargements, *beyond which, however, they were prolonged downwards,* and converging, were fused into dense masses of cicatricial fibrous tissue, connected with ulcer on skin. (2.) CONICAL STUMP from man aged 17. The leg had been amputated at middle third in childhood for disease of ankle. Gradually the stump had become more and more pointed, and one month ago an abscess formed over the end of the tibia, and burst on surface. A frozen section showed that the tibia had grown downwards 1 inch below the lower end of the fibula, so as to protrude the skin, to which it was closely adherent. The fibula was atrophied. The posterior tibial nerve ended in a bulbous enlargement 3 inches above the lower end of the tibia.

V. ORIGINAL COMMUNICATION.

AN ADDRESS ON INTERNAL DERANGEMENTS OF THE KNEE-JOINT AND THEIR TREATMENT BY OPERATION.

By THOMAS ANNANDALE, F.R.C.S. Ed., F.R.S.E., Regius Professor of Clinical Surgery in the University of Edinburgh.

THE general title of this paper is simply employed for the sake of convenience, and because it has been usual to class under this term, first suggested by Hey, certain symptoms in connexion with the knee-joint, the causes of which were not always explainable. Our increasing pathological and clinical knowledge of these and other conditions is, however, leading to a better distinction and treatment of this series of symptoms; and it is with the hope of assisting in this advance that I wish to record the results of my experience.

Three years ago I read to this Society a short paper[1] giving an account of a new operation for the relief of a displaced semilunar cartilage. The good result obtained in this case encouraged me to operate in other cases of knee-joint derangement which had failed to be cured by the usual methods; and having now opened the knee-joint in seven cases, an opportunity has been afforded me for practically studying the actual conditions causing the symptoms complained of. An eighth case under my care, but operated upon by Dr Cotterill, is an addition to this practical experience. I would here remark that these eight cases do not include the several instances in which I have opened the knee-joint for the removal of one or more "loose cartilages."

The eight cases may be divided into three groups:—

1. Displacements of the semilunar cartilages.
2. Growths in the interior of the joint.
3. Growths from the bone protruding into the joint.

I. *Displacement of Semilunar Cartilages.*—In this group there were four cases, three affecting the internal cartilage and one the external.

Case I. (already published).—A miner, aged 30, was admitted on November 1st, 1883, with well-marked symptoms of displacement of the internal semilunar cartilage of the right knee-joint, which had resisted various forms of treatment. My operation for fixing the cartilage by opening the joint, replacing the displaced structure, and stitching it in its proper position by means of catgut sutures, was performed on November 16th. In seven weeks the bandage and splints were removed, and movements of the joint practised. The patient was examined in April 1884, when he was found to be completely cured of his symptoms, and all the movements of the joint were perfect.

Case II.—U. B., aged 32, a gardener, was admitted on November 23rd, 1885. He had suffered from symptoms of displaced internal semilunar cartilage of the right knee for one year, and he attributed his symptoms partly to constant kneeling at his work and partly to a twist of the joint. As ordinary treatment failed to relieve, and as he was unable to follow his employment, the usual operation was performed. When the knee-joint was opened, it was seen that the affected cartilage had been almost entirely torn away from its anterior attachments, and was folded backwards upon itself, lying towards the inter-condyloid notch. It was drawn into position, and stitched there by catgut sutures. The splint was retained for six weeks, when it was removed, and movements of the joint practised. This case also was completely successful, and the patient was seen six months afterwards, when he was actively engaged at his work.

[1] *Brit. Med. Jour.*, April 18th, 1885.

Case III.—J. G., aged 35, a miner, was admitted on July 19th, 1886, suffering from symptoms similar to those in the last two cases, which had existed for about nine months. The knee affected was the right one, and there was no distinct history of an injury. The usual operation was performed on July 21st, and the patient was dismissed, with the splints still applied, on August 24th. He returned at the end of a month, when the splints were removed, and movement of the joint ordered. Six weeks after, he again returned, when it was found that his symptoms were greatly relieved, and the movements of the joint were natural, except that he could not flex the knee to quite the full extent; otherwise the limb and joint were perfectly right. In this case the internal semilunar cartilage was found to be only partially separated from its anterior attachments.

Case IV.—U. U., aged 28, a soldier, admitted November 4th, 1886. Six months before admission he was running quickly down some stairs, when he fell, his left knee receiving a severe twist. He was carefully treated in the military hospital for several weeks, and at the end of this time he could walk a little, but only with the injured limb in the straight position. When he tried to bend the knee, he felt something "give" in the joint, and it became locked, but by a little manipulation and movement of the joint, he could relieve the symptom. There was tenderness over the position of the external semilunar cartilage, and in certain movements of the joint the cartilage could be seen and felt to be displaced. The operation for replacing and fixing the cartilage was performed upon November 11th, and he was dismissed with the splints retained upon December 13th. He returned at the beginning of January of this year, when the splints were removed, and movement of the joint ordered. On February 2nd the patient was walking with a strong and useful leg. The movements were not yet perfect, but the old symptoms had entirely disappeared.

The conditions found in these four cases confirm the following facts, which have been already more or less recognised by surgical authorities:—

1. That one or other of the semilunar cartilages—most frequently the internal one—is liable to be displaced, and to cause more or less interference with the movements of the knee-joint.

2. That this displacement may be slight—as is most common—or severe, and that the amount of displacement depends upon the extent of separation of the attachments of the cartilage.

3. That it is the anterior attachments of the cartilage which are most frequently separated.

In my former paper, I expressed the opinion that the displacement might take place suddenly, as in the case of a twist or wrench of the joint causing some separation of its attachments, or by a more gradual separation, as in the case of certain employments, especially

those in which much kneeling is required; and I still think that this opinion is correct.

Mr Henry Morris (Holme's and Hulke's *System of Surgery*, vol. i.) uses the terms "median" and "marginal" displacements of these cartilages. In an important paper upon this subject (see *Edinburgh Medical Journal* for December 1886 and February 1887) Dr Scott Lang, from a thorough and sound study of the anatomy of the knee-joint and its movements, endeavours—and, in my opinion, succeeds in his endeavours—to explain how and why the semilunar cartilages are displaced, and he further strengthens his views by references to cases clinically observed. He specially points out that displacement of the internal semilunar cartilage may occur when the leg is rotated outwards, and that when the external semilunar cartilage is displaced, it is from rotation of the leg inwards. Accordingly, he suggests as an assistance to the treatment of this injury, that when the internal cartilage is affected, the patient should keep the toes directed inwards, and in the case of the external cartilage, the toes should be kept outwards.

In all four cases, the diagnosis of the condition was arrived at, before the operation, by a study of the symptoms, which were those usually described as characteristic of this accident; but I have met with other cases in which it was almost impossible to say positively what the exact condition causing the symptoms was. Fortunately, a careful exploratory incision into the joint in doubtful cases which are causing trouble, and which resist other treatment, will, as a rule, not only determine the cause, but admit of its removal. Perhaps the most important consideration in connexion with my reported cases is the successful treatment of them by operation. This is a distinct advance in connexion with the treatment of displaced semilunar cartilages; and I think that, from the experience of these cases, I am justified in advocating the operation which was described in my former paper, always provided that the condition is seriously interfering with the comfort and usefulness of the patient, and has failed to be cured by milder measures.

Let me briefly state the steps of the operation. An incision is made along the upper edge of the tibia, on the side corresponding to the cartilage displaced, and it should extend from the border of the ligamentum patellæ outwards or inwards, according to the cartilage affected, for a distance of about three inches. The tissues having been divided, and the synovial membrane exposed, all vessels should be secured before the joint is opened. This having been done, the synovial membrane is incised in the same direction as the external wound, and the parts examined. A blunt hook is then inserted, and hooked round the anterior margin of the displaced cartilage, which is in this way brought into its proper position, and held there while two or three interrupted catgut sutures are passed through it and the periosteum and fascia, over the edge of the head of the tibia. In this way the cartilage is

firmly secured in its proper place. The edges of the external wound are then brought together by sutures, and the dressing and a splint applied.

II. *Growths in the Interior of the Joint.*—In this group there were three cases, two of them examples of fatty growths, the third of myeloid sarcoma.

Case V.—Miss ——, aged 21, came from England to consult me in January 1886, on account of an affection of her right knee. About one year before she had twisted the joint, and ever since had suffered more or less from pain and stiffness in it. Several slight attacks of synovitis had also occurred in the same knee, and had been treated by rest, counter-irritation, and support. No permanent relief having been obtained, she sought my advice in regard to the joint. On examination, little was to be seen externally; there was some fulness on the inner side of the ligamentum patellæ, near the position of the internal semilunar cartilage, and there was also tenderness upon pressure over this point. The joint could not be moved freely, and the patient complained of a "catch" when she tried to bend or extend it. The condition made her limb weak, and prevented her from taking any active exercise. The case had many of the symptoms usually present in displacement of the internal semilunar cartilage, but they were almost constant, and there was no very decided locking of the joint. As she urged me to do something to relieve the condition, and as other treatment had failed, I suggested an incision over the position of the semilunar cartilage. This incision was made in the usual way; and, upon opening the joint, it was found that the cartilage was in its proper position, but that a small mass of fatty and fibrous texture was lying over the inner and anterior margin of the cartilage where the fulness existed. This growth was connected to the synovial membrane, and was movable, and could be drawn forward. It was the only abnormal condition observed, and it was evidently the cause which interfered with the free movements of the joint. Having seized the growth with forceps, I drew it forward, and stitched it to the periosteum on the upper edge of the tibia, so as to fix it, and prevent its passing between the joint surfaces. The wound healed well, and she sat up at the end of a fortnight. She was advised to begin careful movements of the joint in another week; but as she was very sensitive to pain, and did not carry out my instructions thoroughly, I gave her chloroform a month after the operation, and freely moved the joint. After this she made rapid progress, and in another month was able to walk well. Since then she has married, and she recently wrote me that since two months after the operation, her knee has been perfectly well, strong and free in all its movements.

Case VI.—M. P., aged 40, admitted on March 27th, 1886.

About a year before admission, he fell and hurt his left knee, and ever since the joint has been painful, and liable to constant interference with its movements, the patient describing the condition as a sudden "locking" of the joint. Externally, the joint appeared normal, and no displacement of the semilunar cartilages could be detected. Having to be out of town, I asked Dr Cotterill to take charge of the case, and he, on April 26th, made an incision, as in the former case, and, on opening the joint, found a condition very similar, except that the growth was larger. Having ligatured the base of the growth with catgut, the greater part of it was cut away, and the stump stitched to the periosteum and fascia, over the margin of the tibia. The wound healed without trouble, and the splints were removed and movements of the joint ordered on June 10th. He returned to show himself last December, when the result was seen to be perfect as regards the movements of the joint.

Case VII.—Mrs D., aged 33, admitted on February 11th, 1886. Eighteen months before admission, she began to suffer from occasional pain in her right knee. Sometimes she could walk well, but at other times the joint was stiff and its movements interfered with. On examination, a movable body could be detected upon the outer side of the joint, close to the position of the external semilunar cartilage during some positions of the joint. Pressure and manipulation caused the body to disappear.

On February 16th an incision was made along the line of the external semilunar cartilage, and the joint was opened. This cartilage was found in its proper position, but, close to it and attached to the synovial membrane, was a small tumour, the size of a horse bean. The neck of the tumour having been ligatured with catgut, the growth was removed, and the wound in the joint closed. Microscopic examination proved it to be a well-marked example of myeloid sarcoma.

On March 14th, the wound being healed, she was allowed to begin movements of the limb; and, shortly after, she left the hospital with good movement in the joint.

These three cases, in two of which the condition was attributed to injury, are good illustrations of the condition of tumour in the knee-joint. They further illustrate that such a condition may seriously interfere with the movements of this joint; and, again, they illustrate the good results obtained by a careful operation for their removal.

The symptoms met with in these cases, although in some points resembling those caused by a displaced semilunar cartilage, were sufficiently distinct. The symptoms produced by a growth in the interior of the joint will probably vary according to its mobility, size, and position. If the growth has a loose or long attachment, allowing it to change somewhat its place, the symptoms present

will scarcely differ from a case of movable cartilage, which retains some attachment to the synovial membrane; but, when the growth is more fixed and causes inconvenience, as in the two first cases related, the characteristic symptoms will be that the stiffness and interference with the joint movements are constant or almost constant. Also, there will generally be a fulness at some point of the joint, and the position of this fulness is likely to be most frequent at one or other side of the ligamentum patellæ.

In regard to treatment, I think that the experience of these three cases, taken with that of several recorded cases presently to be referred to, justifies my opinion that these growths should be removed by operation when the condition is seriously impeding the joint movements, or causing painful symptoms, and when ordinary treatment has failed to give relief. In advising an operation, it must be understood that no other disease of the joint exists. An exception to this is simple synovitis, which is often present in all the conditions described in this paper, and which need not interfere with the operation. A little point connected with the removal of these growths is that experience proves it is only necessary to remove the loose portion of the growth, if its nature is simple; or it may even suffice, as in Case V., to draw it away, when small, and secure it with a stitch, so that it will not pass between the joint-surfaces, and interfere with their free movements.

In the *American Medical Record* for June 26th, 1886, there is an interesting paper on this subject by Dr R. F. Weir. Dr Weir records two cases of his own and one of his colleague, Dr Bull, in which tumours were removed from the knee-joint. In one case, the tumour was composed of "vascular connective tissue, rich in fat and connective tissue cells." In the second case, the growth was a "fibro-sarcoma," and in the third the growth was a "giant-celled sarcoma." Weir also refers to other cases published by Simon, Volkmann, König, and Lauenstein. Barwell (*International Encyclopædia of Surgery*) mentions that he removed two fatty growths from the knee, one being situated upon each side of the ligamentum patellæ. He expresses the opinion that some of these growths are formed in the subsynovial tissue, and gradually bulge into the joint. Volkmann, under the term "lipoma arborescens articulorum," has described a condition of multiple fatty growths having their origin in connexion with the fringes of synovial membrane.

III. *Growth from the Bone protruding into the Joint.*

Case VIII.—Miss H., aged 55, was seen with Dr P. A. Young and Dr Gibson of Portobello, at the beginning of last year. She had suffered for several months from pain and stiffness in her right knee, and latterly she found that the movements of the joint were

interfered with by something "catching in the inside of the joint." An examination showed slight chronic synovitis, and upon the outer edge of the patella a small hard body was felt, apparently firmly attached to the synovial membrane. But owing to the synovial thickening, it was difficult to decide whether or not it was connected to the bone. In the movements of the joint the edge of the patella came in contact with this body, causing great pain. After consultation it was agreed to remove this body, and I accordingly made an incision parallel to the outer edge of the patella, and opened the joint. It was then seen that the body was an outgrowth from the articular surface of the femur, and was firmly attached to the bone. By means of a chisel the base of the growth was cut through and the body removed. It was an outgrowth such as is so commonly met with as a result of chronic rheumatic arthritis, and was half an inch in length and about a quarter of an inch in diameter at its base. The patient made a good recovery; the wound was soundly healed at the end of a month, and in three months she was able to walk well, and was much improved. During the winter she has suffered from further attacks of rheumatic inflammation in this joint, which has caused some pain and stiffness, but she is able to use the joint and limb usefully, although there is still a little "jerking" in it.

It is well known how commonly outgrowths of chronic arthritic origin cause interference with the movements of this and other joints, and it cannot be said that such cases are very favourable for operative interference; but still I think that, in cases where a single and distinct growth is causing much pain or stiffness, the question of an operation may be taken into consideration, and the case just recorded proves that the removal of such a growth may be successfully performed, and may also, even if not perfectly relieving the symptoms, improve them. It is not an operation that I would wish to urge very strongly, and the general condition of the patient, the condition of the joint, and his or her wishes—the question having been properly explained—would influence me in advising its performance.

Lastly, in opening the knee-joint for the removal of growths, the incision will be best made over the position of the growth, if it can be felt. Should its exact position not be determined, and the case be one suitable for operation, I would suggest that the incision advised for the fixing of the internal semilunar cartilage be employed, as it was found very convenient in the three cases reported.

Dr Haddon demonstrated, and said the practice of the manipulations recommended by Wharton Hood for such cases had earned him (Dr Haddon) a considerable reputation as a bonesetter.

Dr Duncan said it must not be supposed that the method of manipulation devised by Hutton and described by Wharton

Hood was to be put in practice in every case of derangement of the knee-joint. He had seen a good many cases in which it had been put in practice with most disastrous results. Not only so, but the tearings of the semilunar cartilage which took place might be greatly aggravated by wrong movements of this sort, and want of success often followed the operation. The patient often came to know the proper method for doing this in his own case better than either bonesetter or surgeon. Mr Annandale's paper was one of very great value, as showing the possibility of curing a great many cases which had hitherto been always difficult to treat. There were a considerable number of people going about with conditions of the knee-joint which it was not easy to describe, the result of injury, or as Mr Annandale put it, the result of strain on the joint. The paper showed that a certain number of them, where there was no palpable loose body, might be cured by opening the joint. Many were to be met in other ways; sometimes by apparatus, which might have to be of an elaborate character, meant to prevent the joint doing certain movements, such as that of rotation. Recently he saw a gentleman having both joints in this condition, in which he hinted the propriety of interfering surgically; but the patient was not prepared to submit, as he was kept comfortable by an apparatus. A remarkable fact in connexion with these cases was the tender spot which existed midway between the internal lateral ligament and the inner side of the patella just over the internal cartilage. As to the bodies known as loose cartilages, the direct method of removal was the most satisfactory, and was practised by most of them. He had had a number of varieties. The last one reminded him more of the exostosis of the distal phalanx of the great toe, with cartilage at one end and bone at the other, than anything else. Many of them had no cartilage at all.

Dr Cotterill said the pain in these cases arose from the loose body being locked between the joint surfaces, and not because it was merely loose. It should be remembered that the semilunar cartilages in the healthy joint had a considerable range of movement, moving backward in flexion and forward in extension. The locking of the joint always took place when the cartilage, being abnormally loose, was carried too far back in flexion, and then got nipped between the bones in the act of extension. The fatty or fibrous tumours (upon one of which he had operated) were in reality an overgrowth in the alar ligaments, which were placed on each side of the subpatellar synovial cushion. The function of these alar pads and cushions were (in conjunction with the posterior and lateral ligaments and other parts) to prevent over-extension of the knee-joint, by coming in between the bones on full extension. When the alar pads were hypertrophied, this action was, so to speak, anticipated or exaggerated, with the result that the joint became locked. Dr Cotterill did not in the least agree with the

remark made by one speaker that the manipulative proceedings described were originated by bonesetters, or restricted to their practice. He had known them, used them, and taught them for several years. As he knew it, the method in dislocation of the internal cartilage was to fully flex the thigh on the abdomen and the leg on the thigh, to turn out the foot, and then quickly and thoroughly to extend the leg and thigh. This method he had frequently used, and almost always with complete success,—having, amongst others, completely relieved Dr Scott Lang himself of a dislocation of the internal cartilage. In the cases where permanent success had attended these manipulations, it was presumably due to the fact of a limited synovitis being set up by the movements; and this had sufficed to glue the cartilage into its proper place, when it had been reduced by the manœuvre described above.

Dr Scott Lang stated, with reference to luxations or subluxations of the semilunar cartilages, that Dr Cotterill and Dr Haddon had been successful in the immediate reduction of such. The manipulations which they described, however, ought to be founded on exact anatomical knowledge. He noticed a similarity between the manœuvres recommended by Dr Haddon and Dr Cotterill. Dr Haddon struck the knee violently on the outer side of the joint; Dr Cotterill's plan was to rotate the tibia outwards. Now, each of these manœuvres would have the effect of separating the internal condyle from the inner portion of the head of the tibia, and so of freeing the internal semilunar cartilage should it be held imprisoned there. This was probably the most common form of luxation, and hence the frequent success of the treatment. There were cases, however, to which it was wholly inapplicable. For example, there was a dissected specimen preserved where the external cartilage had been entirely torn from its surrounding attachments, and stood vertically between the condyles. There were other cases where the external cartilage became displaced only when the knee is completely flexed. Such cases as these required totally different treatment. He had found on experimenting on the cadaver that after having got a cartilage jammed between the femur and tibia, violence used in a wrong direction might have a very injurious effect. Taking hold of the foot, one possessed great leverage, and the cartilage could be split or crushed and severely damaged between the femur and tibia. Regarding the use of retentive apparatus, he wished to bring one practical point before the Society. It was this, supposing the internal cartilage to be the one affected, the tibia ought to be kept as much as possible rotated inwards, in order to obviate recurrence of the luxation. Should the external cartilage have been affected, rotation outwards would procure a corresponding safety for it. This was palliative treatment, but like the so-called palliative treatment of hernia, it might be also curative if well and carefully applied.

This treatment was based upon both anatomical and clinical evidence. Anatomically it could easily be seen that when rotation of the tibia was made inwards it was done by the semitendinosus, gracilis, and sartorius on the inner side of the knee-joint, and the inner portion of the head was inevitably at the same time brought into close apposition with the internal condyle. The internal semilunar cartilage was thus securely held in place. In rotation outwards the biceps acted in a similar manner as regards holding the external cartilage safely in its place between the external condyle and the outer part of the head of the tibia. In some fragmentary writings it would be found stated that so-called "internal derangement of the knee-joint" occurred when the foot was everted by the toe striking a stone. Now, it was distinctly traceable that this statement invariably referred only to luxation of the INTERNAL CARTILAGE. This coincided in a remarkable manner with the anatomical point above referred to. But further, he could in his own person testify to the practical value of this hint; and he had, from the knowledge of it, been able to give valuable advice to others. One of his fellow-students, however, now in practice, had a subluxation of the external semilunar cartilage, and in his case the knee felt much safer and stronger when the foot was pointed outwards. In fact, he had reproduced the luxation by crossing one leg (the affected one) over the other and turning the toe inwards. He thought these facts explained the chief, if not the only points which could be attended to in the construction of retentive apparatus to prevent the recurrence of such luxations.

Dr Caverhill described a case in which he had succeeded by manipulation in enabling a man to walk home a distance of about six miles after being brought in a cart suffering from what was called dislocation of the knee-joint.

The President asked Professor Annandale if he could tell anything of the future history of the case of myeloid sarcoma?

Professor Annandale said he presumed the patient was well, as if anything had occurred he would have heard of it. He agreed with Mr Duncan that most surgeons had met with cases which had come under the notice of Mr Hutton, and there was a good deal of humbug about them. The bodies called loose cartilages might come from one of three sources—the synovial membrane, deposits within the joint, or portions of articular cartilage knocked off. He should like to direct attention to the paper of Dr Scott Lang, who had not done himself justice that night. He had carefully studied, on anatomical and physiological grounds, the mode of displacement of those cartilages, and had shown them the proper movements to make for their reduction.

Special Meeting.—February 16, 1887.

Professor GRAINGER STEWART, *President, in the Chair.*

ON EMPYEMA.

By WILLIAM RUSSELL, M.D., F.R.C.P.Ed., Lecturer on Pathology in the School of Medicine; Clinical Medicine Tutor in the Royal Infirmary; and Physician to the New Town Dispensary, Edinburgh.

WHEN asked by one of the Secretaries to contribute a paper to this Society, the subject of Empyema was suggested to me by the case I presented at our last meeting. I have had some difficulty in deciding how to treat this subject. I thought first of simply detailing the cases I have personally been responsible for, and making a few remarks on the general subject; but on second thoughts I decided to treat it in a more general way, and not only to utilize, regardless of their chronological order, the cases which I myself have had, but also to make use of the experience I have acquired by having, in one way or another, been brought into personal contact with many cases for which, however, I was in no degree responsible.

With reference to the general question of effusion, either serous or purulent, into the chest, I wish to refer to the physical signs which are present in children when the effusion is moderate or scanty. These signs are particularly indefinite. The local dulness is slight, and may be overlooked, and often passes unrecognised, because of the vigour with which percussion is usually performed. In these cases it is necessary to percuss with extreme lightness, and the sense of slightly increased resistance, which is present, is as valuable as the pitch and quality of the sound, if not more so. The normal type of breathing is present over the affected side, or it may be slightly softer than the normal puerile. The respiration over the unaffected side, on the other hand, is exaggerated, and in the more marked cases might readily enough be mistaken for bronchial. The degree of exaggeration depends, of course, on the greater or less amount of effusion causing a proportionate amount of work to be thrown on the unimpeded lung. The possibility of the healthy lung in a case of moderate pleural effusion being mistaken for a pneumonia has, I think, to be recognised. The indefiniteness of the signs, and yet the importance of appreciating such as are present, have long been impressed on my mind. I have had many cases in which a very slightly increased dulness, and a slightly increased sense of resistance, have led me to diagnose effusion in children when the stethoscope gave me little help. Such cases I have seen dragging out a very unsatisfactory course, until I removed the small quantity of fluid which I believed to be present. After the

removal the cases rapidly recovered. The same difficulty in diagnosis is present when the effusion is purulent and scanty. In illustration of this I may give the following case, which, owing to its having been complicated with capillary bronchitis, strikingly illustrates what I have stated to be the character of the physical signs when effusion is slight.

Case I. was a child thirteen weeks old. I saw it on 28th May 1882. It was evidently pained, and the breathing was much accelerated. The right half of the chest was slightly dull, but the respiratory sounds were audible all over it; not only that, but râles were audible even to the base posteriorly. I suspected fluid, and explored with the hypodermic syringe, and withdrew it full of pus. With the aspirator I then withdrew 4 or 5 ozs. of pus. This relieved the infant, and the dulness was diminished; but there were crepitations over both lungs. The child was suffering from capillary bronchitis in addition to the empyema, and in spite of all I could do for this condition, the lungs became crowded with fine crepitations, the cyanosis deepened, and the infant died on the 30th, two days after I had aspirated.

The case which I presented at the last meeting further illustrates this; the facts are as follows:—

Case II.—Emma T., aged 8½ months, was brought to me at the New Town Dispensary on the 1st September 1886, and was diagnosed as a pleuro-pneumonia of the posterior part of the right base. Mr Hope Grant, one of the students attached to me at the Dispensary, got the case in charge, and I may at once express my appreciation of the conscientious and intelligent perseverance he displayed in its care, for it was largely owing to these qualities in him that the successful result which you have seen was attained. On the 14th September 1886 he asked me to see the child with him, as it had become much worse. On examination I diagnosed pleural effusion on the right side. There was dulness, but the respiratory sounds were perfectly audible, while over the unaffected side the type of respiration approached the bronchial, owing to its extreme harshness and the prolongation of expiration. The child was cyanotic, its pulse small and irregular, its breathing rapid, and its aspect one of suffering and danger. I punctured, and withdrew a hypodermic syringeful of clear serum. The following day, although the child seemed to have improved in its general condition, there was no alteration in the physical signs, so I aspirated, but only got about 2 ounces of clear serum. After this the child seems to have got on pretty well for a time, but on the 30th of September I again saw her, and withdrew 4 ounces of thin pus, or what I may call purulent serum. I again saw the child on the 4th of October, when she was looking better, although she was sweating occasionally, and was emaciated. The

right half of the chest was dull from the mid-axillary line backwards. Breathing was audible all over this dull area, but it was only one-half or one-third as loud as over the unaffected side. When I saw the child a week afterwards, on the 11th October, the dulness had apparently increased, and the child's general condition had continued most unsatisfactory. Breathing was still audible all over the affected side. I again explored, and withdrew a hypodermic syringeful of thick, greenish pus. I opened into the empyematous cavity the following day, making two openings and passing a drainage-tube between them. At the operation about 5 ounces of thick pus were evacuated, and on exploring the cavity I found it to be encysted. The drainage-tube was removed on the 18th of November, and the local dressing was stopped on the 8th of December.

The next case is an example of a common enough type in youth:—

Case III.—Thomas H., aged 15, was on the out-patient list of the Carlisle Dispensary. I saw him on 1st March 1881 with the house-surgeon, under whose care he was. The boy was suffering from great dyspnœa. There were the usual physical signs of a very abundant effusion into the left pleural cavity, the most incontrovertible of the facts in evidence of its abundance being that the heart was beating in the line of the right nipple. With reference to the history of the case I can say little; no systematic thermometric observations were kept; the attack dated from eleven days before I saw him, and there had been profuse perspirations. He said he had been subject to pain on the affected side for years. The indications imperatively demanded immediate aspiration. At the operation I removed fully 90 ounces of pus. The pus was perfectly sweet. This was on the 1st of March. On the 6th there was evidence of reaccumulation. This being verified by means of the aspirator, the lad was put under chloroform and the pleural cavity opened. On the 10th, three days after operation, it was noted that the patient was perspiring much less, the tongue was clean, the appetite good, the drainage was perfect, the heart's impulse was slightly to the left of the sternum, and the pleural cavity was being washed out daily. I have no further note until the 16th of April, a month and ten days after the operation, when it is stated that the patient had been doing well, that the cavity had become small, and that he was only to be dressed every second day. There is a further note to the effect that he had had occasional pains in the right hypochondrium accompanied by sweating, but these attacks had always passed off without any special treatment. Although I have no note of the precise date when the wounds were completely healed, I believe they were in between six and seven weeks from the date of the operation; at all events they were so on the 31st May, when I noted that the lad looked plump, that there was

considerable flattening, and that there was practically no respiratory movement on the left side. The percussion note was impaired all over the affected side. At the end of inspiration there was a subcrepitant crackle, which was more marked over the upper than the lower half of the lung.

Case IV.—This case was recorded at length, and published in the *Glasgow Medical Journal* for September 1883. I will, therefore, only briefly sketch its course here, although it was one of great interest, and belonged to the important group of Primary Acute Fœtid Empyemata.

Helen T., æt. 21, was seen by me on the 1st of September 1881. She complained of pain in the left side, which dated its onset from the previous day, and was aggravated by inspiration and the movements of the trunk. There was slight cough. The temperature was raised and the pulse quickened. The lower part of the left half of the thorax was kept almost motionless during respiration. There was no dulness over this region, but the respiratory murmur was exceedingly faint, save when the patient hesitatingly drew a longer inspiration, and cautiously relaxed the rigidity of the part. My belief at that time was that the girl had an attack of diaphragmatic pleurisy. During the first week of her illness the temperature, which was taken night and morning, ranged from 101° to 103° F. The pain was during all that week not only more severe than I had ever seen pleuritic pain, but also more obstinate and unyielding. Hot poultices gave practically no relief, and morphia hypodermically gave only temporary relief. Friction was at no time heard, but there was evidence that fluid was slowly accumulating.

On *8th September* I explored with the aspirator to ascertain whether the effused fluid was purulent or not. The result was that a pint of excessively fœtid pus was withdrawn, the fœtor filling the room in which we were. I did not empty the chest, as it was evident that any measures short of incision and free drainage were not to be thought of. The following day I made two openings into the pleural cavity, and passed a large drainage-tube between them. The wounds became covered with a dirty gray false membrane, and the patient's general condition denoted a degree of septic absorption, for she had profuse sweatings and some slight rigors. The pleural cavity was daily washed out freely with a 1 in 80 solution of carbolic acid, and oakum was used as a local dressing. The discharge under this treatment became daily less fœtid, and before a week had passed it was quite sweet, and had diminished much in quantity.

On *3rd October* I noted in my case-book that the discharge had gradually lessened in quantity, and that a day or two before this date the drainage-tube had been withdrawn from the upper wound, as there seemed to be only a sinus connecting it with the lower

one. The patient's general condition was fair, there were neither nocturnal sweats nor intermittent sensations of chilliness; cough, however, had frequently been very troublesome, and was at this date severe, hard, and paroxysmal in character, and the patient's mother had noted a bad smell with her breath for some days. On the suspicion that there might be a small encysted accumulation of pus, I made two exploratory punctures with the aspirating needle in the situation which seemed most likely after careful physical examination, but with a negative result.

On 8*th October* the lower wound had contracted so much that it would no longer admit the drainage-tube.

On 12*th October* the cough was again severe; the breath had a fœtid odour when the cough was violent, and she had perspired much the two preceding nights. A probe was passed along the track of the sinus which still remained, but without result.

On 13*th October* she expectorated about half a teacupful of purulent matter tasting "like rotten eggs." The cough abated after this, and the breath lost its fœtor.

On 23*rd October*—that is, between six and seven weeks after the operation — the wounds were quite healed. Breathing was audible all over the side which had been affected, except in the neighbourhood of the cicatrices, and there was no retraction. I did not see the patient again until the 26th of January of the following year. She had been in good health in the interval, but for two days at this date she had a severe cough, with a purulent and bad smelling expectoration, and associated with nocturnal sweatings. These symptoms had been preceded by pain in the epigastrium and upwards in the sternal line. The symptoms again disappeared, and when I saw her on the 21st of February she had continued well, and had gained in both strength and flesh.

The problem which so prominently presented itself in this case was: How came the empyema to be primarily fœtid, seeing there was no evidence of pulmonary gangrene or of communication with the outer world? Soon after this case had occurred Professor Gairdner and Professor Buchanan, of Glasgow, published jointly a similar case, and, strange to say, it was the first of its kind which Professor Gairdner had seen. I looked up the subject, in a very incomplete way I admit, but I got together eight cases, including my own, and found that they had all occurred on the left side, and at least one of them had, like my own case, been diagnosed as a diaphragmatic pleurisy. I suggested at that time that the phenomenon might be explained by the proximity of the large intestine to the diaphragm on the left side, and that the contamination had probably been aided in its transit by the inflamed condition of that muscle. However that may be, the fact remains, that a primary empyema on this side may be fœtid. I have not looked further into this subject, and should be glad to know if any undoubted case had occurred on the right side, for if there had,

the anatomical relations of the parts would not be a sufficient explanation.

Case V.—The notes of this case I have either lost or mislaid. The patient was a girl of about 14 or 15 years of age. She had for a time been under the care of a medical friend who had first seen her, when what appeared to him to be the ordinary symptoms of advanced phthisis were present, the leading symptoms being copious purulent expectoration, hectic, sweating, and emaciation. Soon, however, his attention was drawn to a swelling in one of the intercostal spaces below the level of the right nipple. He then suspected the true nature of the case, and asked me to see it. I may briefly say that the case turned out to be an empyema discharging through a pulmonary fistula, and pointing at the spot on the anterior aspect of the chest already referred to. I opened it at this point, but it soon became evident that the opening did not drain the cavity. A second opening was accordingly made at the lowest possible interspace posteriorly. I at the same time explored the limits of the cavity, and found it to be a very large one, extending far up, both anteriorly and posteriorly. With the establishment of free drainage the girl rapidly improved, lost all hectic symptoms, became plump and fat, and was going out. So far as I can remember the wounds healed, and she was regarded by her friends and her medical attendant as practically cured, although I confess to having been sceptical of the completeness of the cure, owing to reasons which are partly apparent and others which need not be specified. She had a relapse, I think, but I left the district and saw no more of her, and I have neglected to acquaint myself with her subsequent history. This case hardly requires any comment. The possibility of such an error in diagnosis is apparent. Its cure I have already expressed my scepticism about, for the case had lasted long enough for the lung to have become firmly bound down in its compressed state, and the pus cavity was of such vast dimensions that I regarded the case, from the first, as one in which excision of ribs would probably be ultimately necessary.

Case VI. was a boy aged about 4 years, whom I saw with Dr Murphy of Carlisle on the 1st of May 1882. He had had measles three weeks before I saw him. There were present the ordinary signs of fluid in the left chest. The breath sounds were absent. The heart was beating at the right nipple. There was no bulging of the intercostal spaces and no local œdema. The temperature was elevated, and he was and had been perspiring profusely. Respiration was accelerated. With the aspirator I drew off about a pint of greenish pus which was quite sweet. The heart returned to its normal position, and the respiratory sounds became audible over the back. There was soon evidence of reaccumulation, but the parents would not sanction any further operative interference.

On the 27th they at last consented; but when I saw the child he was cyanotic, with a pulse which was at times imperceptible, and so ill that we considered operative interference to be hopeless, at least, it seemed to be so certain that the child would die in our hands if we attempted the operation, that we, what is commonly called, *funked* it. Looking back on the case, and with the courage of wider experience, I do not think that in similar circumstances I would hold my hand, but would face the risk of the opprobrium which is apt to accrue from death occurring during medical interference. The case was a desperate one, and required the courage desperate cases demand.

These six cases are, in their respective ways, examples of common enough types. I have still, however, a few more facts connected with cases to refer to. I well remember the case, which occurred a number of years ago, of a man who died without any more definite opinion of his case having been reached than that "there must be pus somewhere." At the post-mortem examination I found to my chagrin that neither I nor others had discovered a localized empyema confined to the upper part of the lateral region of the right half of the chest, that is the axillary region. Of course there were difficulties, but difficulties can hardly be pleaded as excuses.

Another case was that of a man with a patch of gangrene at the right base, followed by pleurisy and effusion. Above the limit of the effusion there was a very markedly tympanitic note which led the physician in charge of the case to insist, in spite of all the evidence I could lay before him, on its being a pneumo-thorax, the result of perforation at the seat of gangrene. The post-mortem examination revealed a pleural cavity full of pus.

This tympanitic note above pleural effusion is as interesting as it is common, and need not be discussed now, but it is evidently possible for it to mislead.

Among the unfortunate sequelæ I have seen after operation I may mention pyæmia. A very disappointing case, which I saw in a friend's hands, was that of a lady rather advanced in life, who had been operated on, and had practically recovered, when the wounds, which were not quite skinned over, were attacked with a very virulent form of erysipelas, which rapidly proved fatal.

Passing now to the differential diagnosis of purulent from serous effusion, it seems almost superfluous to say anything, as it is so easy by means of the hypodermic syringe to remove all possibility of doubt. Among the symptoms, however, which point to the effusion being purulent may be enumerated, profuse perspirations appearing early in the history of the case; the local temperature being higher on the affected than on the unaffected side; severe and obstinate pain;[1] and, as some believe, the presence of œdema of the chest wall. If pus is suspected, the sooner the question is settled absolutely the better for the patient.

[1] This symptom I learnt from my friend Dr Lockie of Carlisle.

Coming now to treatment, we have to bear in mind that cases are on record where, especially in children, aspiration of the whole or even a part of the pus has been followed by recovery. I have not had this experience so far, but it has to be reckoned with. Instead, however, of attempting to examine the wide controversies which occupy the pages of medical literature, I may perhaps be allowed to state what has hitherto been my general line of procedure. It is usually some days after the onset of a pleurisy before one suspects pus, and if the effusion is not great, the suspicion is reserved for perhaps a week or so, the length of time depending on the strength of one's suspicions. If pus is found, I am careful to remove it as completely as possible by means of the aspirator; this requires time, for spasmodic cough is apt to come on and to necessitate the interruption of the operation. After this, if there is evidence of reaccumulation in the course of a few days, the chest ought to be again explored; and it is well to be ready to operate, for if pus is reaccumulating, the sooner a free outlet is provided for it the better.

With reference to the operation itself I have little to say. I am not a surgeon, but this is one of the operations it is desirable a physician should be able to do, as surgical skill is not always available at the proper moment. I therefore have a very simple way of performing it. I pass the needle of the aspirator or the hypodermic syringe into the cavity. Taking the needle as my guide I cut through the soft parts into the cavity and enlarge the opening freely with a probe-pointed bistoury. I then pass a strong probe—indeed for this purpose I always use a uterine sound with its normal bend—into the wound and downwards until it reaches the diaphragm; I feel for the first intercostal space above it, and press the point of the sound until I feel it from the outside; I cut down on it, and enlarge the opening as I did the upper one. I tie a piece of strong string of some kind round the point of the probe and withdraw the probe with the string attached; by means of this string a thick drainage-tube is introduced into the pleural cavity, one end allowed to project from either wound, and united externally by means of a stout string. By this very simple proceeding thorough drainage is established; but, what I think of great importance is, that complete control is obtained over the drainage-tube, which can be taken out and washed and reintroduced at pleasure and without difficulty. The cavity can be very thoroughly washed out by filling it from the top wound and keeping the lower one closed, and then allowing it to empty itself by the lower. I have found that the cavity gradually contracts until only the track of the drainage-tube is left, the tube is then withdrawn from the top wound, and the sinus gradually heals from above downwards.

With reference to the further operation of removal of ribs, which is necessary in some cases, I have little to say—the operation belongs exclusively to the surgeons. I have seen a considerable number

of cases in which it was performed; some were successful, many were not. What I have not yet had sufficient evidence to satisfy my mind on is, whether this operation would ever be necessary if cases came under observation early enough, and if proper steps were taken with them when they did.

If an effusion is left until the whole or a large part of the lung is compressed, and has developed a new fibrous covering fitting its diminished size, it is absolutely hopeless to look for expansion, and it would not only be necessary, as Mr Duncan humorously but truly suggested some time ago, to take a slice off the entire half of the thorax, but it would be necessary to remove the whole half, only leaving a slice.

I have, gentlemen, taken the liberty of submitting my own experience to you on this important subject, and I only hope that the personal form which it bears may be approved by you.

Dr Caverhill showed two cases, and made some remarks on the treatment.

Dr Duncan, called on by the President, said he had not understood he was expected to address the Society, and had not been careful to prepare any special observations, but he should be very glad to lay before them what experience he might have, and all the more readily because Dr Caverhill must have almost led them to believe that surgeons were ready to descend on every case of empyema, and take away a slice of the thorax. Such was hardly the way they looked on empyema. They were inclined to regard it as they did an abscess in any other part of the body. It was to be treated on exactly the same principles. The chief and first rule they applied to all abscesses, with very few exceptions, was, that whenever they diagnosed pus they should proceed to evacuate. That being so, the only question, having ascertained the case to be one of empyema, was how to evacuate. There were three ways in which this might be done. One was by aspiration, another by simple drainage, and a third by resection of ribs. In his opinion each of these had their value in connexion with empyema. What was the value, in the first place, of aspiration in connexion with abscess? They were coming to look upon aspiration with, on the whole, less and less favour in cases where there was distinct pus. For the withdrawal of thin fluids it was extremely effective, but with some forms of pus it could not be performed. One of the first points in the treatment of abscess by aspiration, which must be universally admitted, was that it was the rarest possible thing to bring about a cure except by repeated aspirations. In a certain number of cases by repeated aspirations they would succeed. In psoas abscess, for example, he had done so by four or five aspirations when combined with careful treatment of the cause, and here a question came in which should be worked out by the physicians—What was the cause of empyema? When they had

removed the cause of the pus in empyema there might be a cure by aspiration. But there was another important point in connexion with aspiration which made it advisable that in every case it should be first resorted to, and that was, that by repeated aspirations they might bring about adhesions from the lung surface to the chest wall, and so encyst the empyema. This was of importance, both in reference to drainage and other methods. In a certain number of cases, therefore, aspiration did good. But supposing aspiration has failed, they came next to drainage, to the making of a simple incision, and that was a thing they would do without hesitation. He would not detain them with any discussion upon the spray, on which his sentiments were tolerably well known, merely saying that Dr Caverhill's single case did not to his mind demonstrate its usefulness as a germicide in the air. The drainage should be as complete as possible, and the pus made or kept aseptic. How was this to be done? First, there was the question Dr Russell had raised as to whether there should be one or two openings. For himself he had come to the conclusion that one opening was sufficient. The only advantage they got from two openings was in late cases where sinuses had formed. Where the incision should be made was another point. Mr Miller had pointed out that to make it too low was a mistake. In the adult falling in would generally take place unless the empyema was encysted. The diaphragm would then rise, and the low opening would become a long sinus, and another opening would have to be made higher up. Therefore it was better to make it at least two inches above the diaphragm, a little below and to the front of the angle of the scapula. There was another point of importance, and that was when the chest collapsed (as it would always at first, though it might rise afterwards) the india-rubber drainage tube had its calibre diminished by the pressure of the ribs, and, causing even absorption of the bone, produced a great deal of pain. In order to avoid this, he always now cut out a portion of one rib, and inserted the drainage tube through the hole thus made. These seemed to be the lines on which they went in drainage. In a very large proportion of cases it was successful. In all young persons he should say, and in all that were kept aseptic, they would succeed, and the lung would expand. In all cases in which the empyema was encysted, and in all cases got sufficiently early, they would also have a cure. But there was a certain number of cases in which they would not succeed. There were some that would not be cured by drainage, and died of amyloid degeneration—those long-continued cases in which there were adhesions by fibrous membranes of such density and thickness that it was absolutely impossible that the lung could ever expand. He was afraid they would never get to the Utopia where all cases of empyema came to the proper people at the proper time. In those cases that became septic, even if got early, the proportion in which drainage failed

was greater than with others. In those long-continued cases in which the falling in of the chest wall had taken place to its utmost extent, and the lung was bound down, they must save from death, and the measure which was of value was certainly resection of the ribs. It was thought some years ago that about three ribs might be resected. He did this, but gave it up, as it was never of any value. Not less than six, and probably eight ribs (the third to the tenth inclusive) should be resected. It could be done by one incision, giving room to remove three to four inches in the middle; above and below rather less. He had done this in six cases, in two of which the operation had to be repeated. Three were cured, two were alive with sinuses (one of them going on well in Fifeshire). The sixth died of pyæmia. She went home with a drainage tube, and unfortunately had been careless about it, taking out the pin that fixed it, and allowing it to slip into the thorax. She delayed to return, not coming back for years, and died the day after her arrival. In all there was a considerable bending of the wall of the thorax. Dr Caverhill had referred to another point—the effort to expand the lung, which would be of value early in a case. There were various ways in which this might be done. He had tried it by putting a sucker on the chest wall, passing a tube through it into the thorax, and connecting it by syphon with antiseptic fluid in a vessel, which could be raised or lowered at pleasure. A very slight lowering had a very considerable effect, sometimes causing great pain from the amount of suction caused. This would act effectually for some hours, and then a little air would get in by the side of the sucker, stopping its action. How far it was useful he did not know. He had not had a sufficient number of cases, and the case in which he had tried it was of very old standing—the case that died of pyæmia. He had thought of trying something valvular—a valve falling up and down with the respiration. But, undoubtedly, the method of suction presented great practical difficulty.

Dr Affleck said the subject of empyema had engaged the attention of the profession since any one had practised scientific medicine. In the writings of the Father of Medicine considerable reference was made to it. Hippocrates was the first to practise auscultation, and he thought he could distinguish between the presence of pus and of serum in the pleural cavity. He was wrong, no doubt, but there were some observations he made on empyema and on fluid in the chest, both as regards diagnosis and treatment, which were of extreme interest, and showed that they had not made such great advance after all on his theory and practice in this matter. They had yet much to find out regarding the causation of empyema. They knew that it occurred sometimes simply from the intensity of the inflammation in persons previously apparently healthy. In others it appeared to be due to the occurrence of complications, as acute fevers. In the case he showed at

last meeting, the empyema had not come on till the pleurisy had been complicated by scarlet fever and erysipelas. With regard to the presence of pus in the pleural cavity, as Dr Russell said, they had an infallible means of diagnosis in the exploratory puncture. This should be practised by every one, and he should be ready to aspirate at the moment. With regard to the symptomatic evidence they had not very precise information. Sometimes the fever, as Dr Russell said, indicated it, hectic symptoms, the appearance of the patient, the exhausted look; but sometimes they would find these present even where there was nothing but serum. He had tapped a large number of times, and sometimes had found serum where he expected pus, and pus where he expected serum. He did not know whether any one had noticed a peculiar character of the cough. He had sometimes noticed a peculiar ringing or metallic sound in it, even when there was no opening into the lung from the pleural cavity. It was worth observation. Probably they would find a difference between sounds of coughs in various thoracic diseases. As to treatment, he agreed with Dr Duncan as to the propriety of trying first aspiration. A good deal depended on the condition associated with pus in the pleura. If phthisis was present they might pursue one line of treatment; if it was not, then another. A good deal depended on the acuteness or chronicity of the case. If Hippocrates did not practise aspiration and drainage, he did something very like it. He treated empyemata in a very interesting way. He sometimes punctured in an intercostal space. He first watched for critical days, and on certain days he interfered; and he (Dr A.) thought it was on the fifteenth day after the fever that he punctured the intercostal space, or bored a small hole in a rib, and let out the matter. He allowed this drainage to go on for ten days, then he poured in a mixture of warm oil and wine, and closed it in by a small piece of linen rag, to which a thread was attached, drew out the rag and evacuated twice a day for ten days, then put in a metal sound, taking it out once or twice a day, and in that way got some very successful results. It was interesting to note this, considering how scanty his anatomical knowledge was. He thought aspiration should be practised in all acute cases in the first place. It should be performed as low as possible, and in that way the lung would probably descend if not bound too much by adhesions, and so obliterate or greatly diminish the pus-discharging cavity. One aspiration sometimes did for children, and even for adults.

Mr A. W. Hare referred to the probably tubercular origin of pleural inflammations in cases where there were no obvious meteorological agencies at work, nor the presence of contiguous inflammations. He recalled to mind the specimen of tubercular pleura shown by Dr Woodhead at the meeting on the 2nd of February, and he cited the recent researches of Keloch and Vaillard in support of his opinion. The latter observers concluded,

from a series of exhaustive observations (see *Archives de Physiologie* for 15th August 1886), that pleuritic inflammations, whether serous, hæmorrhagic, or purulent, were associated with the local development of tubercles, and were therefore to be numbered among tubercular affections. Among other interesting observations detailed in the paper was that of the microscopical appearances in cases of recovered pleurisy. Here the pleura was found studded over with old tubercles enclosed in fibrous tissue and undergoing degeneration, as has been described in other situations. Mr Hare could not accept *in toto* the sweeping conclusions of these observers, but thought them of such importance as to demand careful attention and further investigation. In regard to the surgical treatment of empyema, Mr Duncan had already accurately defined the present state of opinion on the subject, and had sufficiently safeguarded the surgeon from a charge of rashness in cases where it was deemed expedient to remove a large section of the thoracic wall. At the same time, very considerable spinal curvature and thoracic deformity resulted from Estlander's operation when segments of so many as eight or nine ribs were removed. This was not only distressing to the patient, but might predispose to other diseased conditions. Just now this seemed a rather fashionable operation,—perhaps more so than could be absolutely justified, when its results were compared with those obtained from resection of a part of one rib and thorough drainage. The speaker had no desire to disparage the operation in cases where other means had been patiently tried without success; but even in these the question had to be settled, whether the resulting deformity were not worse than the condition prior to the operation. In regard to the operation generally, a clear definition was wanted of where justifiable and useful operative interference ended, and where needless mutilation began.

Dr James, in expressing his appreciation of Dr Russell's interesting and valuable paper, desired specially to state his approval of the concluding remark, viz., that it was to be hoped that fewer and fewer cases of empyema would pass out of the hands of the physician into those of the surgeon. At this the surgeon need not take umbrage, as he believed that in the great majority of cases this transference, so common at present, meant that medical treatment had not been properly directed and carried out. To understand the treatment of empyema one must understand the physical conditions of the respiratory organs. In the to and fro movement of the lungs against the costal pleura in respiration they had a good example of how Nature's acts might at one time tend to do harm and at another to do good. They tended to cause a spread of a pleuritic process, but effusion having occurred, the surfaces having been separated, and the inflammatory process having been allayed, they tended to cause expansion of the collapsed lung. If the chest wall were unopened, both inspiration and expiration

tended to cause this expansion of the lung, but if there were an opening, expansion would be brought about mainly by expiration. Coughing would specially tend to this, but every expiration would aid, and they could often in such cases observe that patients, as it were, instinctively expired with closed glottis, and driving air from the sound lung into the collapsed one promoted its expansion. How successful such efforts could become many of them had seen. If, however, they could obtain expansion without a drainage opening into the pleura it would, no doubt, be better, and a mode of treatment which he had followed in empyema was repeated tapping. If in such cases they tapped at intervals of two or three or four weeks they did no good—the fluid simply reaccumulated, and the lung got more and more firmly bound down; but at intervals of two or three or four days the lung got a chance of expanding, and as the fluid which was drawn off often seemed to lose to some extent its purulent character and become more watery, the absorbents took a share of the good result. Of course cases might occur where this plan might fail, but he had often seen it entirely successful. When an opening had to be made for drainage, the plan of excising a small portion of rib, as carried out by Dr Duncan, seemed to be a particularly good one; it precluded closure by approximation of the ribs, and rendered the use of any rigid structure (which always caused pain) unnecessary. As regards the position for the opening, he believed that the fifth intercostal space, about the mid-axillary line, would be the best. This corresponded with the centre of the dull area which cases of absorbing pleurisy showed, bounded above by the well-known *curved line*. It was low enough down for the fluid, high enough up for the rising diaphragm, and far enough forward to permit of lung expansion from behind. On the subject of resection he would only say, that if it had to be done it should be done thoroughly. If the lung would not come to the chest wall, the chest wall must be allowed to go to the lung. The last point which he wished to mention concerned the character of the cough in purulent as distinguished from sero-fibrinous collections of fluid in the thoracic cavity, as referred to by Dr Affleck. He believed this might be due to the fact that in purulent collections the intrathoracic pressure was usually much higher, and that the expiratory effort required for coughing would be correspondingly interfered with.

Mr A. G. Miller said that he agreed with Dr Duncan that they had not yet begun at the beginning of the treatment of empyema, seeing they had not yet found out the cause. He had seen the remark of an American surgeon, "No organisms, no pus." But Dr Caverhill had said to-night that careful investigation had failed to find organisms in his case. On the other hand, they had heard that tubercle bacilli were always present. Mr Miller had noticed that in those cases in which he had removed portions of ribs they

were much thickened on the inner surface. Had bacilli been present in the pleura he would have expected caries, and not simple hypertrophy, from the irritation of the inflamed pleura. As to the question of when ribs should be removed in cases of empyema, he thought that when the lung was no longer capable of expansion by itself, and was tied down by a thick layer of fibrous as well as granulation tissue (as Dr Duncan had said), that then resection of portions of a sufficient number of ribs to bring about collapse of the chest wall was called for. In such circumstances it was necessary to bring the chest wall to the lung. In this way adhesion of the lung might be obtained, and when once this had occurred, it was possible for the lung, in the course of time, to expand by adhesion to the chest wall in the way so well explained by Dr James, and thus for the cavity to be obliterated. He referred to a case he had published, and showed the preparation where, after removal of portions of several ribs, the chest wall had fallen in, the lung had become adherent, and the cavity obliterated at that part all but a small sinus. Had the patient lived long enough, or more portions of rib been removed, the cavity might have been closed entirely, and a limited amount of function restored to the lung. Dr Duncan had referred to him (Mr Miller) as having been one of the first to point out that the rising of the diaphragm after draining an empyema acted as a valve, and occluded the opening if made very dependent at first. He considered it due to Dr Bramwell to say that the possibility of this had been pointed out by him. In the case already referred to Dr Bruce had discovered at the post-mortem that the diaphragm had risen as high as the fourth rib. In concluding his remarks, he would like to mention what he had noticed in some cases of empyema in emptying the chest. At first the pus flowed freely through the drainage tube which had been inserted, then the fluid came in gushes, alternating with rushes of air, into the cavity of the thorax, the pus coming out only to the extent to which the air passed in. This continued for some time, the discharge getting less and less. A time came when the pus was quite below the level of the opening, and no more could flow out if the patient lay still. Then at this moment (he had noticed) the patient suddenly became breathless and cyanotic. This lasted for only a few seconds, and then passed away. He was inclined to think that these symptoms were caused by the full pressure of the atmosphere being brought to bear on the pericardium and heart, and at the same time impeding the expansion of the healthy lung.

Dr Haddon was understood to advocate early tapping in cases of pleurisy with effusion.

Dr Leith Napier, considering the shortness of the time at each individual's disposal, would regard the subject wholly from a practical or clinical aspect. He would remind the Society that he could not be accused of undue temerity in recommending and

carrying out operations within the thorax. In 1878 he published some cases in the *Lancet*, and advocated the emptying of phthisical cavities, and at the same time related a very good instance of the benefits attending free incision in empyema after unsuccessful aspirations. This he ventured to recall, because he now wished to say that he believed that many large serous effusions were rendered purulent by injudicious indiscriminate operative procedures. The absorbent power of serous surfaces was very great. He had frequently seen very large quantities of fluid occupying both sides of the chest become perfectly absorbed without aspiration,—operation being delayed to permit improvement in the general condition. To obtain general constitutional improvement was often the best and safest way of promoting satisfactory cure in pleural effusions, more particularly in children. Another objection to improper or insufficient operation was that should a sinus remain after aspiration they frequently found carious ribs and amyloid degeneration of viscera as sequelæ. The presumptive diagnostic signs of an empyema were—the length of time a copious effusion had existed, a large quantity of fluid with a rapid filling of the chest, rigors and low fever in course of a pleurisy. Afterwards they might have hyperpyrexia. Yet he had refrained from operation in several such cases, and found the ultimate condition to be better, although convalescence might be more tardy than in like cases subjected to surgical interference. It must be remembered that a lung which had become compressed by fluid for a long time, and was airless, would not regain its original condition to any great extent; and that they had change of bony parts, retraction of ribs, falling of shoulders, etc., after the fluid had been removed either by absorption or aspiration, more probably after the too rapid removal by aspiration, when the whole of the fluid was attempted to be withdrawn. The treatment of empyema in children and in adults must be regarded from different aspects. Some authorities taught that aspiration should be performed two or three times before the radical operation of incision. Fräntzel considered the latter always necessary except in tubercular cases, which he treated solely by tapping. On the one hand, Aufrecht had never seen good from simple tapping, even in children. On the other hand, Branthome averred that puncture alone would cure children. Dr Napier's experience agreed with Mr Godlee's, who, in the *Lancet* for 1886 (vol. i. pp. 51, 95 *et seq.*), stated that some cases of empyema in children recovered well without surgical interference. In chronic phthisis a true empyema might be best left alone. In lung abscess, unless it were absolutely clear that the case was tubercular, or when the abscess was in direct contact with a bronchus, he would counsel incision after exploratory aspiration in the case of adults, and, with less confidence, the same in children. Drainage should be maintained for a fortnight, not longer, in children, and in adults one must be guided by the quantity and

condition of discharge. He believed in one opening, and thought that when ribs were removed they should also remove the periosteum—otherwise, especially in young children, if such an operation were really a necessity, the growth of new bone would be probably too rapid. Speaking of empyema in pregnant and puerperal women, he would express the decided conviction that aspiration was quite insufficient, and therefore unwarranted. If any operation was adopted, which must always be considered a very grave question, free incision would give far better results, accompanied by no more, if as much, risk. He must refrain from entering on the question of empyema *sine* micro-organisms; but if Prof. Ogston's researches on the relation of micro-organisms to healthy abscesses were borne in mind, it would be difficult to realize an empyema, a collection of veritable pus, without some organism as cause or result.

Dr William Hunter directed attention to the part played by the diaphragm in promoting the absorption of pleural effusions, the very excess of fluid present in large effusions, by limiting the range of movement of the diaphragm, tending to interfere with their absorption. It was probably this fact which in part explained the marked effect sometimes produced in such cases by the removal of even a relatively small quantity of the fluid, the greater freedom of movement then permitted enabling the further absorption of the fluid to be completed spontaneously.

Dr Watson Campbell, Duns, said the subject of discussion to-night was of special interest, inasmuch as it brought the physician and surgeon on common ground; and he thought there could be but one opinion as to the way in which it had been opened by Dr Russell. As to the diagnosis of empyema, Dr Russell, after mentioning other means of diagnosis, said that the exploring needle was the most certain and satisfactory, and in this he was followed by Dr Affleck. He did not suppose that there could be the slightest doubt about that, but he failed to see that it was, in most cases, of importance to have recourse to it; for, after having ascertained the nature of the fluid, something further must be done at once, and the aspirator would enable the operator not only to determine whether it was serum or pus he had to deal with, but to remove it; it initiated and completed the operation. Then, with reference to the time for surgical interference, Dr Duncan remarked that he treated empyema as he did a common abscess: when pus was present he let it out.

Dr Duncan.—I did not say I would wait for the formation of pus.

Dr Watson Campbell said—Well, he thought aspiration should be done at an early stage. The late Prof. Miller used to teach that there were three areas in an ordinary abscess—a central, and a second and third outside of it; that serum was effused into the first, which degenerated into pus, and that this involved the sur-

rounding tissues when seeking a way out; and that by cutting early and deeply, so as to reach the central area, the degeneration into pus was anticipated, and a way made for escape of morbid material with the least destruction of tissue. Now, he thought something like this mode of procedure should be carried out in dealing with the serous effusion of pleurisy. He believed Dr Haddon was right in advocating that aspiration should be performed early, and that by doing this many cases of serous effusion might be prevented from degenerating into empyema. If the effused fluid were copious, and attended with dyspnœa and visceral displacement, he thought it safer to aspirate than wait in the hope of absorption taking place, seeing that, by delay, there was risk of the fluid changing its character. He had not had much experience of empyema—in fact, he had only had one case—but he had aspirated frequently; and he believed he would have had a greater experience of it if he had delayed aspirating some of his cases, even for a short time. As the case referred to was interesting, he would briefly give the leading particulars. The patient was a young woman. After serous effusion had been going on for about eight days, he thought it would be proper to aspirate on his next visit, if there did not appear to be any arrestment of the effusion. Unfortunately, he was unable to see her for two days, and then he was struck by the change in her appearance. She had an anxious expression, a hectic blush, and was sweating, which led him to suspect degeneration of the fluid. He might here state that, before perforating, he usually made a very slight incision in the skin to facilitate the passage of the trocar and cannula; but he did not remember whether or not he did so in this case. He remembered, however, that the instrument went through the chest wall with a plunge—much further than he meant it to go; so far, indeed, that he believed it went right into the lung. A very violent fit of coughing ensued and continued some time, though he withdrew the trocar and cannula at once into the pleural cavity. After subsidence of the cough he drew off 20 ounces of thick yellow pus; but, during the process, he had to stop several times on account of recurrent cough. After aspirating this quantity he was compelled to desist, as the cough became violent and persistent. Immediately after withdrawing the cannula she began to expectorate pus in large quantity. The cough and expectoration continued night and day for four or five days, gradually, however, decreasing in severity and quantity. The result was satisfactory. He aspirated about the middle of May. By the term-day, the 26th, she was so far well as to bear removal a distance of nine miles, and in autumn was strong and well, and able to engage in out-of-door work as a farm servant. He might mention that she soon after married, that she had four or five children, and enjoyed excellent health.

Dr Byrom Bramwell said that at that late hour of the evening

he would not attempt to consider the whole subject of empyema in any detail; but there were one or two points which had arisen during the course of the debate to which he would like to refer. The fact which Dr Russell had brought forward that in children the breath sounds are often pretty distinctly heard over portions of the pleura which contain fluid was, he thought, a fact of great importance, and one which he could confirm by personal observation. The point was brought under his notice many years ago (either in 1868 or 1869) by the late Sir Robert Christison, and he had frequently satisfied himself that in some cases it is a fact.[1] He had been very much interested in Mr Hare's statement with regard to the French observations of the relationship of the tubercle bacillus to the etiology of pleurisy and empyema, and he thought the point was one which would repay investigation by those who had the opportunity of pathological work. Doubtless, some cases of pleurisy and empyema were due to tubercle, but he very much doubted whether tubercle was the usual cause of these conditions; for his own part, he would certainly hesitate to accept the conclusions to which Mr Hare had referred until they had been fully confirmed by subsequent and independent investigators. He had for long thought that one reason why empyema was more common in children than in adults, was due to the fact that in some cases of empyema in the child the empyema was due to broncho-pneumonic patches on the surface of the lung, the inflammatory condition beginning in the lung and subsequently extending to the pleura. He could not agree with Dr Leith Napier's view that the diagnostic puncture was better avoided. Certainly he himself had seen a simple pleurisy become an empyema after aspiration; indeed, in two cases he had, he thought, produced empyema in this way; but these cases occurred many years ago, when strict antiseptic precautions were not so generally used as they are now. Of recent years he had not seen any such results, and he entirely agreed with the position which Dr Russell had taken up as to the necessity and usefulness of the diagnostic puncture. He thought that it was in many cases the only certain means of determining whether the fluid in the pleura was purulent or not, and rendered other means of diagnosis unnecessary; and he believed it to be a perfectly safe means of procedure. It must, however, be remembered that the hypodermic needle did not clear up all cases. In some the fine needle got choked up with lymph, or the pus was so thick that it would not flow through it. In such cases the instrument should be withdrawn, washed out, and again inserted, or a larger tube used. Time did not permit him to refer to those cases in which a solid intrathoracic tumour was complicated with pleuritic effusion or empyema. The diagnosis in such conditions

[1] The case to which Sir Robert Christison particularly referred was that of a patient living at the south side of Edinburgh, if my memory serves me, whom he had seen in consultation with one of the Keiths.

was often most difficult, and was in many instances materially simplified by means of the hypodermic syringe. The mode of operation in empyema (*i.e.*, when the fluid within the chest was shown by the diagnostic puncture to be purulent) depended, he thought, upon circumstances. His own experience certainly agreed with that of those who say that in the adult aspiration is useless. He had seen cases of empyema in children cured by aspiration, but no such cases cured in an adult. Possibly this was, as Dr James had suggested, due to the fact that the aspiration was not sufficiently frequently repeated and sufficiently thoroughly performed. He, however, doubted that explanation, for in most cases of empyema in the adult (in whom the chest wall is rigid and does not so readily collapse as it does in the child) it is difficult or impossible to draw off all the pus, or anything like all the pus, by simple aspiration; and in order to effect a cure, the complete removal of the pus is most desirable. Any one who had had much experience of aspirating the chest, even, in simple pleuritic effusion, where the lung was usually much more capable of expanding than in cases of empyema, must acknowledge that it is often impossible to withdraw anything like the whole of the fluid; he himself had repeatedly seen such violent constrictive pain, cough, marked cyanosis, and alarming symptoms occur, that the aspiration had to be suspended long before all the fluid was withdrawn. He very much doubted, therefore, whether in the adult empyemata would be likely to be cured by repeated aspiration. Until recently he had always taught that, while aspiration should be tried in the child, free evacuation should be practised under strict antiseptic precautions in the adult, a drainage tube inserted, and the case treated as a large internal abscess. There could, however, be no doubt that a free opening into the chest wall, by allowing the entrance of air into the pleural cavity, and by equalizing the atmospheric pressure on the inner and outer surfaces of the lung, did materially interfere with the expansion of a collapsed lung, and should therefore, if possible, be avoided. He was disposed to think that continuous drainage by syphon action through a tube placed under water was, theoretically at all events, the best means of treatment. By this means a continuous suction can be exerted upon the pleural cavity; the serious symptoms due to rapid aspiration are avoided, atmospheric air is not allowed to enter the pleural cavity, and the atmospheric pressure on the bronchi and interior of the lung is therefore free to act unopposed as a dilating agent; there is not the same risk of septic mischief being set up in the cavity of the pleura. Further, by varying the size of the tube or the length of the syphon, the suction force can be easily regulated. Dr Bramwell was disposed to think that in the adult continuous drainage by syphon suction should first be tried; and if this did not succeed, a free opening should then be made into the cavity of the thorax, and a drainage tube inserted, the strictest antiseptic precautions being

of course adopted. He quite agreed with Dr Duncan that there were cases in which, in consequence of the lung being adherent, and incapable of expansion, all these methods failed, and in which it was advisable to resect the chest wall. In his opinion the risks of this operation were less than the risks which the patient necessarily ran in cases of this description of waxy degeneration and other complications.

Dr Brakenridge agreed with Dr Byrom Bramwell in thinking that many cases of empyema arose from the rupture of small catarrhal-pneumonic-phthisical cavities on the surface of the lung into the pleural cavity. This applied to the disease in the adult as well as in the child. Because this was a possible cause in many cases of empyema, the very greatest caution was necessary in the employment of any negative pressure on the lung within the pleural cavity, or positive pressure within the lung itself, which was calculated to expand the lung on the diseased side. By the injudicious use of the aspirator in connexion with an exhausted jar, such powerful traction was often exerted upon a diseased portion of lung that a small opening on the surface was thereby converted into a free communication between a bronchial tube and the pleural cavity, or a communication was formed which did not previously exist, and a pneumo-thorax thus established. This result unquestionably followed aspiration in the case of a servant girl, aged 17, who was brought into Dr Brakenridge's ward on 4th December 1885, suffering from what appeared to be an attack of simple pleurisy. The girl's family history was markedly phthisical, but she had enjoyed good health until three days prior to admission. She was hanging out clothes to dry, and caught cold. This was followed by pain in the left side, and when she was examined three days later, she presented all the signs of pleurisy with effusion. That day, as the fluid was accumulating rapidly and giving rise to urgent dyspnœa, 32 ounces of cloudy fluid were removed by the aspirator. This was found to contain a small amount of pus. The operation gave considerable relief. Two days later the fluid, having again accumulated, was again removed by aspiration to the amount of 38 ounces. The fluid on this occasion contained a good deal of pus. Soon after this second tapping it became clear that the upper half of the pleural cavity contained air, and that a free communication had become established between a tolerably large bronchial tube and the pleural cavity near the lower angle of the left scapula posteriorly. Soon the patient began to expectorate large masses of pus, and great displacement of the heart took place, the apex beat being in the right anterior axillary line. The temperature and pulse rose, and she became very breathless and hectic. A few nights later she was nearly suffocated with the quantity of pus which poured through her lungs into the trachea; and, as her life was in danger, Mr John Duncan, at Dr Brakenridge's request, laid open the pleura by free

incision in the eighth interspace in the left axilla, inserted drainage tubes, and dressed the wound antiseptically. A free evacuation of the pus was thus effected, and from that moment the patient's expectoration gradually and rapidly diminished, until in a few days it had entirely ceased. In a similar manner her temperature, pulse, and respirations steadily fell. Adhesion of the pleural surfaces took place gradually but quickly from above downwards, and the lung expanded so perfectly that a few weeks later no difference in size could be detected by the physical signs between the lungs on both sides, and that on the left side appeared equally healthy with that on the right. The apex of the heart, which before the operation was beating in the right anterior axillary line, after recovery beat in the normal position. The patient still—two years after the operation—enjoyed perfect health. This case illustrated well the danger of aspiration, and the excellent results which might follow a timely free incision. A very interesting question arose at this point. By what mechanism was the expansion of the lung effected in such a case? It was quite true that (as Dr James had suggested) some degree of expansion must be effected by the lung being pressed downwards and forwards by the expiratory efforts and the fixing of the lung by pleuritic adhesions at the advanced positions it had so reached. This would not, however, explain the perfect expansion of a lung in communication with the external atmosphere through the pleural cavity. In a clinical lecture which Dr Brakenridge gave on the above and several other cases two years ago, he stated his belief that the force of the right ventricle acting on the inner surfaces of the pulmonary vessels must be allowed to have a considerable influence in gradually expanding the collapsed lung. This followed from Pascal's law that "pressure exerted anywhere upon a mass of liquid is transmitted undiminished in all directions, and acts with the same force on all equal surfaces, and in a direction at right angles to their surfaces." As the lung, gradually expanding from above downwards, became fixed at certain levels, the pressure within its vessels would drive the next portion a little further down, and so on from point to point until a considerable degree of expansion had been effected. The suggestion of Dr James that expiratory efforts on the part of the patient must tend to expand the collapsed lung was most important. Certainly, just as emphysema was produced by the forcible expiratory efforts of coughing, so expansion of a collapsed lung might be similarly assisted by voluntary expiratory efforts. Not one, but probably several of these factors together, effected the expansion of the lung in the most satisfactory cases, such as that to which he had referred.

Dr A. J. Sinclair considered what he might term the pneumatic treatment of empyema a most important one, and one to which sufficient attention was not paid. There was no doubt that they were deprived, by having a fistulous opening in the intercostal

wall, of the powerful force of inspiration in reproducing expansion of the lung, but they still had a powerful agent in the expiratory forces. He was glad to find, from Dr James's remarks, that he had not underrated this agent, and in the remarks which had fallen from Dr James he entirely concurred. He desired to relate a case bearing on the point. It was one which happened in his practice some years ago. A young man had been under his treatment for empyema. The case, after being opened, ran an ordinary course, and progressed satisfactorily up to a certain length, when there progress ceased, and an obstinate fistulous opening remained. The man, being formerly an amateur player on the cornet, wearied for a trial of his favourite instrument and asked permission, which was granted. From that date the fistulous opening steadily closed. They had here an instance where the expiratory force acted as a powerful agent in approximating the pulmonary to the costal pleura and contracting the cavity. They might have observed, in Dr Affleck's case, that after the ribs had been resected that the lung partially expanded, and that Dr Affleck directed their attention to the existence of the respiratory murmur at a place before inaudible—showing that all hope of the re-expansion of the lung was not lost. Dr Sinclair also exhibited a drainage tube which he had been using for some years. It was an ordinary rubber tube, with rubber shield, stiffened at its commencement by the introduction of an inch of lead pipe. The advantage of this was the prevention of intercostal space pressing on a flexible tube; and also by keeping the shield a short distance from the skin, antiseptic material might be packed round the commencement of tube, and the discharge drained more into the centre of the dressings. This method prevented the pus escaping between the skin and dressing, and permitted the patient to be kept more comfortable, and less liable to the danger of septic mischief. The tube answered for any large chronic abscess.

Dr John Thomson said he had met with two cases in which pus, at first apparently free in the pleural cavity, after two or three aspirations reaccumulated in the form of a small circumscribed empyema over the base of the lung posteriorly. Out of more than forty cases of empyema in children, which he had the opportunity of observing while Resident to a Children's Hospital, fourteen had been cured by simple aspiration. In the majority of these the pus was small in amount and circumscribed. In some of the cases one aspiration was enough, in most two or three were required, and in one as many as five. He had only seen one case of empyema in a child in which the pus, when first removed, was offensive. It was on the right side.

The President, in calling on Dr Russell to reply, congratulated him on the successful discussion he had inaugurated, and the exceedingly valuable record of cases, and the deductions therefrom, which he had given in his paper at the commencement.

Dr Russell said the discussion had been one of the best he had heard at the Medico-Chirurgical Society. For that he did not take the credit to himself. It was due rather to the interest and importance of the subject. He did not propose to go over the remarks of the various speakers in detail, but would refer to one or two points that had impressed him. As to the differential diagnosis of purulent effusion, the only new point mentioned was the ringing character of the cough which was noted by Dr Affleck and Dr James. With reference to the ætiology two suggestions had been made. One by Dr Hare, quoted from recent French observations, was that pleurisy in every case was a result of tuberculosis. With Dr Bramwell he agreed that in this country they were not as yet prepared to accept a sweeping statement of this kind. It involved the wide question of what tuberculosis really was, both from the clinical and the pathological point of view. Much work had yet to be done at this subject before they could come to a completely satisfactory conclusion regarding it. The other suggestion came from Dr Bramwell, who believed that, in children especially, an empyema was the result of or followed upon a patch of broncho-pneumonia. Dr Brakenridge mentioned a case in which this seemed to have occurred, and in which perforation took place. As to treatment, the suggestion made by Dr James, and the idea suggested to Dr Sinclair by his patient, the cornet player, were of the utmost importance. As to operative procedure, there had been a tendency to advance from the present treatment, and if they could cure by tapping every day, or every second day, they made a distinct gain. Dr Bramwell's idea of endeavouring to empty the pleura by continuous syphon action was an extremely good one. With this idea they were familiar, but it was not much practised in this country. He had stated his belief in the absolute harmlessness of the hypodermic needle or aspirator as an aid to diagnosis, and all the speakers, with one exception, had agreed with him. He entirely disagreed with the statement that with fewer aspirations there would be fewer empyemata. In his own cases only one was serous at the beginning. From Dr Lockie of Carlisle he learned that he had had twelve cases, in only three of which was the fluid serous when first aspirated. There were, therefore, a large number of cases purulent from the beginning; and for himself he would sooner adhere to the old lines, and have the satisfaction of seeing a child rapidly regaining health and strength after incision, than leave it to drag on in a wretched condition, becoming emaciated and hectic, in the hope that the pus would dry up. It was the following out of that line of treatment that led to the special kind of experience hospital surgeons and physicians had in empyema.

Meeting V.—March 2, 1887.

Professor GRAINGER STEWART, *President, in the Chair.*

I. ELECTION OF ORDINARY MEMBER.

The following gentleman was elected an Ordinary Member of the Society:—John Keay, M.B., Mavisbank House, Polton.

II. EXHIBITION OF PATHOLOGICAL SPECIMENS.

1. *Mr A. G. Miller* showed a FOOT amputated for WHITE GANGRENE, and a DRAWING of the same done for him by Mr Caird. The gangrene was due to embolism of the tibial arteries, in the anterior so complete as to cause the dorsum of the foot to become suddenly quite anæmic, and exhibit the pearly whiteness which had been so well brought out in the drawing. Another point of interest was that the parts had been kept absolutely sweet for a period of two months, and when the limb was amputated the gangrenous portion was quite dry and mummified, and the skin at the white part transparent.

2. *Dr James Carmichael* exhibited the BRAIN OF A GIRL, aged 4, who had died in the Royal Hospital for Sick Children from meningitis. The convolutions, it would be noticed, were much flattened, and the lateral ventricles, which were distended with fluid, were considerably enlarged; the foramen of Monro was sufficiently large to admit the little finger. He would like to draw attention to the very unequal dilatation of the lateral ventricles, the left being roughly about twice as large as the right. This condition was seen, although less marked, in the brain of a child which Dr Bruce had kindly shown to the Society for him at the last meeting. At the base would be seen the ordinary signs of meningitis, the pons and a portion of the under surface of the cerebellum being covered with a layer of fibrinous lymph, in which a few gray tubercular nodules were visible. There were also some tubercular nodules in the corpora striata. The membranes between the cerebellum and pons were closely adherent, and the foramen of Majendie apparently closed. Macroscopically no tubercle had been found in any other of the organs, but the mesenteric glands were enlarged and evidently tubercular. The child had been about five weeks under treatment, and the clinical features of the case generally were those of well-marked tubercular meningitis. Symptoms of paralysis and coma did not supervene till the end of a fortnight; previously the only head symptom was persistent vomiting. During the last twelve days the child had remained in a condition of general spastic rigidity, opisthotonic spasm being well marked for two or three days, but ultimately passing off.

III. Exhibition of Instrument.

Dr A. Bruce showed a new form of RADIAL MICROTOME.

IV. Original Communication.

THE DIAGNOSIS AND TREATMENT OF CEREBRAL ABSCESS DUE TO EAR DISEASE.

By P. M'Bride, M.D., F.R.C.P. Ed., F.R.S.E., Surgeon to the Ear and Throat Department of the Edinburgh Royal Infirmary, and Lecturer on Diseases of the Ear and Throat, Edinburgh School of Medicine ; and

A. G. Miller, F.R.C.S. Ed., Surgeon to the Edinburgh Royal Infirmary, and Lecturer on Surgery, Edinburgh School of Medicine.

Part I., by P. M'Bride.

To say that ear disease is a common cause of cerebral abscess is but to repeat a truism—recognised alike by physician, surgeon, and specialist. Were statistics required, we might refer to the elaborate table compiled by Gull and Sutton, and published in Reynolds' *System of Medicine* (vol. ii.), or to Lebert's classical papers on this subject (Virchow's *Archiv*, vol. x.)

Chronic suppuration of the middle ear is the variety of aural disease which is most likely to lead to cerebral mischief. Indeed, exceptions to this rule are rare ; occasionally, it is true, acute suppuration within the tympanic cavity proves fatal, and Zaufal has recorded an example of serous catarrh of the middle ear resulting in death from intracranial inflammation. We may, however, look upon such cases as interesting only from their rarity.

Granted, then, that in chronic suppurative otitis media we have to fear head symptoms, the question before us may be discussed under the following subdivisions :—

1. Given a case of chronic suppuration of the middle ear with head symptoms, what are the pathological conditions which may give rise to these symptoms ?

2. How far can we by their semeiology alone differentiate these conditions ?

3. Will a careful examination of the ear, objective and subjective, aid us in localizing the brain lesion ?

4. Having made our diagnosis as complete as possible, should treatment be attempted ?

(1.) *The pathological conditions which may produce head symptoms in the course of chronic suppuration of the middle ear.*

There is no doubt that in these cases it is often extremely difficult to say precisely when the patient, even though he present acute symptoms, is actually attacked by a gross lesion of the brain. In other words, it is frequently impossible to define when the stage of irritation is over and active inflammation has begun.

To illustrate my meaning I may be allowed to refer briefly to a case which I saw in consultation with Dr Bruce. The patient, a little girl suffering from chronic suppurative otitis media, was suddenly attacked by intense pain in the mastoid region. This part was swollen and tender; the whole aspect, when the patient was examined, was that of acute fever. The temperature was 106°, there was great intolerance of light, and a rigor had occurred. We agreed to open the mastoid antrum next day, and meanwhile ordered leeches over the mastoid. The patient had long hair, and the leeching was entrusted to a relative, a medical student. Owing to an oversight, the leech bites were allowed to bleed all night, the blood being prevented from flowing over the pillow by the patient's hair. Next morning she was almost quite well, and in a day or two made a good recovery. Another case similar in many points, but more grave, because the symptoms developed less rapidly and lasted for some days, is the following:—

A young woman had been seeing me off and on for some years for an otorrhœa, of which she was at one part of this period virtually free. She had a perforated drum membrane, suppuration of the tympanic cavity, and in addition a granulation growing from the posterior and upper wall of the osseous meatus. This granulation was perforated in its centre, showing it to be the pouting orifice of a sinus, which led backwards towards the mastoid. Repeated removal of the growth and cleanliness eventually relieved her of the otorrhœa. After this I did not see her until asked to do so by Dr Carmichael, who considered that cerebral symptoms had set in. The ear was at this time blocked by a large granulation, and discharging freely. The mastoid region was swollen and tender; the granulation was removed, but no benefit resulted, so we agreed that opening the mastoid should not be delayed. I need not detail the steps of the operation. It was found that the mastoid process proper was converted entirely into ivory-like bone. In such cases, speaking generally, it is not justifiable to chisel to any great depth, but in this instance we considered the danger to be so imminent that I determined to go on until the mastoid antrum was reached. This intention was, however, frustrated by an unexpected contingency. While working with a gouge at a considerable depth the wound suddenly filled with dark venous blood, evidently from the lateral sinus. No drain was established; the bleeding was stopped by plugging with lint dipped in carbolic oil, and yet the desired result was obtained. The head symptoms disappeared, and complete recovery was only retarded by a pneumonia—whether embolic or not I do not know. I may remark that the operation completely removed the otorrhœa, and that, *mirabile dictu*, the granulation never reappeared. For this last-named result I can offer no explanation except that the counter irritation of the operation acted as a cure; but then at once arises the question, What is counter irritation?

Of course in these two cases there was probably no severe gross lesion of the intracranial cavity, for one was relieved by bleeding alone, and the other by counter irritation—certainly of a severe kind—and bleeding. In this connexion, however, I cannot but mention that cases have been recorded of optic neuritis, and even paresis of the sixth nerve with head symptoms, occurring in the course of, and due to, ear disease in which recovery took place.[1] From these remarks it will be seen how difficult it is to say when the patient is in a hopeless condition, and when such a serious operation as opening the cranial cavity is justifiable.

I think, however, that, all this notwithstanding, those who are accustomed to see head cases will agree with me that, in all of those which end fatally, a time arrives at which both the medical man and the relatives begin to give up hope, and it is then that any attempt at relief, however heroic, becomes justified. Chronic suppuration of the middle ear may produce various intracranial changes, viz., cerebral abscess, meningitis, and thrombosis of cerebral sinuses.

The form of cerebral abscess commonly met with is that which has been termed by authorities "chronic." Chronic it may be in its pathology, and chronic I often believe it to be in its symptoms, yet when it has advanced so far as to be suspected, the symptoms are commonly acute, and the patient has not many weeks to live. Meningitis, when due to ear disease, seems to be usually of the purulent variety, the pus from the ear bursting through the osseous walls of the cavities of the ear, and detaching, while not unfrequently perforating, the dura mater.

Thrombosis of the cerebral sinuses, especially the lateral, may result from ear disease, but not uncommonly an intracranial abscess is associated with this condition, perhaps as a result of spreading phlebitis.

(2.) *Can we, by their semeiology alone, differentiate these three conditions?*

This question I have no hesitation, from my own experience, in answering in the negative. Pain in the ear and head is the most prominent symptom in a case of chronic suppuration of the middle ear which threatens to have a fatal issue. Rigors are commonly met with; the tongue is either abnormally red or furred; cerebral vomiting may occur; the temperature often rises; but no doubt in many cases of intracranial abscess both pulse and temperature are at some period after the onset of acute symptoms persistently subnormal. Indeed, when this condition exists or has existed, I should look upon its existence as rendering it more probable that a well-defined collection of pus—rather than diffuse meningitis or phlebitis—is the cause of head symptoms. Twitching, delirium, and, finally, coma, may be met with both in cerebral abscess and meningitis, while in both there may be congestive changes in the

[1] Roosa, *Diseases of the Ear*, 6th edition, p. 551.

optic discs. As a rule, marked paralysis, except of the facial, is rarely associated with head mischief arising from chronic ear disease, although I have met with paresis of the third. Paralysis of the facial may signify simple neuritis or carious destruction of the osseous canal. At all events, this form of paresis is generally due to direct action on the nerve trunk as it wends its way through the petrous bone, and not to disease of its centres.

In thrombosis of the cerebral sinuses—a condition not unfrequently associated with both abscess and meningitis—we have also headache as a prominent symptom. Further symptoms gradually develop, and death ensues. In these cases, however, there are certain points to guide us. In the first place, the course of the case corresponds more or less to pyæmia, and metastatic abscesses may develop in other parts. The joints may become tender, although more often the pains are most marked in the course of muscles (Lebert). The fever is of a distinctly remittent type,—the temperature fluctuates, the pulse rate varies, and profuse perspirations and rigors occur at intervals.

If the lateral sinus—and this is the form most to be dreaded in ear cases—be involved, we may have œdema of the mastoid region; but as in such cases there is frequently mastoid disease, this symptom loses much of its value. The inflammatory affection may, however, extend down the jugular, and the latter is then corded and the side of neck tender to the touch, while spasm of the sterno-mastoid muscle may co-exist. It should, perhaps, also be mentioned that thrombosis of the internal jugular may cause symptoms pointing to pressure on the glossopharyngeal, pneumogastric, spinal accessory, and hypoglossal nerves.

So far, then, we may say that to some extent we have means of forming an opinion as to whether we have to deal with intracranial inflammation associated with phlebitis or not. The fact, however, remains, that in most cases—even when we are fairly certain that phlebitis of the lateral cavernous or superior petrosal sinus exists —it is impossible to exclude the co-existence of such conditions as meningitis and abscess.

Again, in cases of abscess we have sometimes a history of previous cerebral attacks recovered from; and if this be clear in the case of a patient suffering from head symptoms associated with ear disease, the physician is inclined to suspect encapsuled abscess rather than diffused meningitis.[1] This, then, leads us to the third question—

(3.) *Will a careful examination of the ear—objective and subjective —aid us in localizing the brain lesion?*

The late Mr Toynbee, in a paper read before the Medico-Chirurgical Society of London in June 1851,[2] laid down certain rules as to the association between different forms of ear mischief and brain disease, viz.:—

[1] Ross, *Handbook of the Diseases of the Nervous System*, p. 626.
[2] See *Transactions*, vol. xxxiv.

1. Affections of the external meatus and mastoid cells produce disease in the lateral sinus and cerebellum.

2. Affections of the tympanic cavity produce disease of the cerebrum.

3. Affections of the vestibule and cochlea produce disease in the medulla oblongata.

Recent writers on cerebral abscess are still in the habit of quoting these statements, and therefore it behoves us to consider them carefully, with a view to ascertaining whether they be accurate or not. That disease of the meatus may possibly give rise to brain mischief I will not deny, but that such cases are most exceptional is well known. Moreover, in the paper referred to there is given an elaborate and carefully compiled table of fatal cases of ear disease, which, however, fails entirely to show that disease of the meatus is likely to cause fatal cerebral complications. If we desire to understand what Toynbee considered to be disease of the meatus we must turn to his text-book, published, be it observed, in 1860—that is, nine years after the paper referred to —and there (page 73 *et seq.*) we find described as "chronic catarrhal inflammation of the dermoid meatus extending to the brain" a case of chronic suppuration of the middle ear involving the meatus and mastoid cells in its course, and, finally, causing phlebitis of the lateral sinus. There was extensive caries of the temporal bone, and on post-mortem examination it was found that the tympanic membrane was absent. Toynbee looked upon this last-named condition as secondary to the inflammation of the meatus, but in the light of modern otology we know that in such cases the tympanic cavity is first attacked.

As to Toynbee's assertion that inflammation of the nervous apparatus of the ear produces disease of the medulla oblongata, it rests upon slender foundation; for of three cases, in one it is not mentioned that the medulla was affected, and in the other two it seems, from his table, that this part may only have been involved in a diffuse purulent basilar meningitis. This view is confirmed by Schwartze, who observed a case of primary purulent inflammation of the labyrinth, in which diffuse purulent basilar meningitis was produced. The case just quoted is almost unique, for generally purulent inflammation of the internal is secondary to a similar condition of the middle ear. It is further confirmed by Bezold,[1] who has analyzed, from a statistical point of view, forty-six cases of necrosis of the labyrinth. In seven cases only did death result, and in most of these it was due to disease of the cerebellum. Toynbee's other propositions, however, as to the connexion between mastoid disease and affections of the cerebellum and lateral sinus on the one hand, and affections of the tympanum and cerebrum on the other, have much more in their favour, as is clearly shown by his tables, although the data are not sufficient to warrant a definite conclusion.

[1] *Zeitschrift für Ohrenheilkunde*, vol. xvi.

It behoves us now to consider whether we have any reliable data to ascertain what part of the brain is most commonly attacked in ear disease. With this object in view, let us glance for a moment at Roosa's statistics of fatal cases of ear disease collected from different sources,[1] forty in number. Briefly analyzed, the results are as follows:—Cerebral abscess, 21 (1 abscess of pons); cerebellar abscess, 8 (in 1 there was also abscess of the cerebrum); diffuse suppuration, 1; clot in lateral or other sinus, 8 (in 1 there was also an intracranial abscess); severe meningitis, 1.

This accounts for thirty-nine cases, and the fortieth is not sufficiently detailed to be of statistical value. The result of this analysis seems to show that more than 50 per cent. of cases of fatal ear disease are due to cerebral abscess, and probably most of these were owing to direct communication between the suppurating middle ear cavities and the brain or its membranes. There is one point in Roosa's tables that surprises me, viz., that there should be so few cases of death from diffuse purulent meningitis recorded, and I cannot avoid the suspicion that purulent meningitis and abscess have been classed together.

In considering a subject of such vital importance, however, we must not trust to statistics compiled by one author, and we therefore turn to the table showing the causes of cerebral abscess compiled by Gull and Sutton.[2] We here find twenty-seven cases in which the condition was due to ear disease, and the localities of the abscesses were as follows:—Cerebrum (1) localized abscess, 19 (1 in pons); (2) diffuse suppuration, 2 (1 between dura mater and bone); cerebellum, 6.

Here, again, we see that the cerebrum is most commonly involved; indeed, is involved in by far the largest number of cases; and I may add that in most of these the middle lobe, or that which is in most intimate relation with the ear, was the part affected (fourteen out of twenty-one).

It has seemed to me (notwithstanding these statistical results) that I am not justified in drawing any positive conclusions from them without compiling a table of my own. I have succeeded in collecting forty-four cases, and have only included in my list examples in which both the ear and brain were studied on the dissection table. I may mention that while post-mortem examination revealed that in by far the largest number of cases both tympanum and mastoid were involved, yet pain and swelling behind the ear were by no means constant clinical features. Hence we must infer that there may be an accumulation of decomposing pus in the mastoid antrum, and yet no symptoms indicating its presence.

Classified according to the part of the ear involved in the suppurative process, my tables give the following results:—

[1] *Diseases of the Ear*, p. 554 *et seq.*
[2] Reynold's *System of Medicine*, vol. ii.

Part of the Ear affected.	Cerebral Abscess.	Diffuse Meningitis almost invariably Purulent.	Cerebellar Abscess.	Thrombosis of Lateral or other Sinuses.
Tympanum and mastoid. Total, 28.	12; 3 with diffuse meningitis; 4 with thrombosis.	6	2; 1 with thrombosis.	8
Tympanum alone, 5.	3; 1 sub-dural; 1 between bone and dura mater.	0	1 with pus over roof of tympanum.	1
Middle ear and labyrinth, 11.	0	6; 1, also a small abscess in the pons; 1 with thrombosis of lateral sinus.	4	1

These data, then, go to prove that in the majority of fatal cases the tympanum and mastoid are involved; and inasmuch as it seems that the mastoid antrum may be involved without clinical symptoms, we may, for practical purposes, consider the cases of tympanic and mastoid disease together with those in which the tympanum seems to have been involved alone.

This gives us out of thirty-three cases, fifteen of cerebral abscess, six of diffuse purulent meningitis, three of cerebellar abscess, and nine of thrombosis of the lateral (or other) sinus. Out of eleven cases in which the labyrinth also suffered, in six there was diffuse meningitis, in four cerebellar abscess, and in one thrombosis.

These facts show that to examine the condition of the labyrinth is of considerable importance in any case in which we have arrived at the conclusion that the brain lesion is a localized collection of pus.

If, then, in such a case bone conduction, as tested by the tuning-fork, be retained on the affected side, the chances are in favour of the abscess being in the cerebrum; but if bone conduction be lost, then we should suspect the cerebellum to be the seat of mischief. Beyond this, I do not think my statistics enable us to ascribe diagnostic value to the condition of the ear. For my own part, I should attach some importance to the presence or absence of mastoid tenderness. While, with or without tenderness, the mastoid antrum may be filled with pus, I am inclined to think that when pain is experienced behind the ear it shows a tendency to increased tension in the antrum. In other words, the inclination of the pus is to go backwards, and involve the cerebellum and lateral sinus; while if there be no indication of tenderness, I should

expect a spread of inflammation through the roof of the tympanum to the cerebrum. Mr Hulke suggests[1] that in the young, brain abscess from ear disease is more commonly situated in the cerebrum, and in adults in the cerebellum. To test the accuracy of this suggestion, I have analyzed all the cases in my table of intracranial abscess in which the ages of the patients are stated, and find a total of nineteen. Of these fourteen were cerebral and five cerebellar. Of the former in three cases only were the patients under 20, the ages being (19, 17, 8); and of the latter one was 11 years old, the others being over 20. Of course the numbers are not large enough to permit of an absolutely definite conclusion being drawn, but they go far to disprove Mr Hulke's hypothesis.

(4.) *Should Treatment be attempted in Cases of Otitic Intracranial Suppuration?*

Although cerebral abscesses due to injury have been repeatedly treated with success, yet those caused by ear disease have generally been allowed to end fatally, without any attempt at interference. So far, I believe, only three successfully treated cases have been published, recorded by Schondorff,[2] Truckenbrod,[3] and Gowers and Barker.[4] In the two former, however, there was an external fistula, the surrounding scalp being œdematous and tender. In the last-mentioned case, an abscess of the temporo-sphenoidal lobe was correctly diagnosed by Gowers, and successfully operated upon by Barker. In this instance the point of the trephine was inserted $1\frac{1}{4}$ inches behind, and the same distance above the meatus. Before proceeding to operate on the bone, the state of the opening for the mastoid emissary vein was examined; for Mr Barker believes that, where the suppuration is behind the tentorium on the surface of the petrous bone, pus will escape through this foramen. I doubt the accuracy of his conclusion, for it seems to me that this view can only hold in cases of diffuse cerebellar suppuration, but not in localized abscess.

In a very interesting and suggestive paper,[5] Mr Hulke advocated opening otitic intracranial abscesses, and although his cases were unsuccessful, and although in them an examination of the ear was not carefully undertaken, either during life or after death, yet his observations are of the greatest value. This author rightly calls attention to the importance of low temperature and pulse as a diagnostic of abscess, as opposed to meningitis, in cases of ear disease with cerebral complications; in all his cases (three in number) both temperature and pulse were subnormal. Unfortunately, however, cerebral abscess may co-exist with high fever. In Dr Gower's case this was so, but at the beginning of the disease pulse and temperature were abnormally low. I would, therefore, combining these facts with my own experience, lay considerable stress, in any given case, on the fact that, whatever the present

[1] *Lancet*, 3rd July 1886.
[2] *Archiv für Klin. Chir.*, 1884.
[3] *Archives of Otology*, 1886, operation by Schede.
[4] *British Medical Journal*, Dec. 1886.
[5] *Lancet*, 3rd July 1886.

condition, the pulse and temperature had, after the onset of acute symptoms, been at any time subnormal.

Hulke in one case found pus in the cerebellum, and to reach this part safely, he perforated the occipital bone "two centimetres behind and inwards from the mastoid process, the crown of the trephine encroaching on the inferior curved line. This spot," he goes on to say, "was selected as being sufficiently beneath the lowest level of the horizontal part of the lateral sinus, and also sufficiently removed inwards from the descending part of the sinus. The dependent portion of the spot was also thought advantageous for the drainage of the abscess, if found. The spot was easily uncovered, by raising the scalp with a horse shoe incision, the ends of which lay slightly above the level of the external occipital tuberosity, and the bend slightly below the level of a line joining the apices of the mastoid processes. The muscles were divided by a cut down to the bone parallel to the superior curved line, and, together with the periosteum, peeled down to a sufficient extent with a raspatory. The inferior curved line easily felt proved a good guide to the selected spot. The severed occipital artery was easily secured with a pressure forceps and afterwards tied."

This, then, seems the best method of reaching the cerebellum for operative purposes, and although we have seen that the differential diagnosis between diffuse meningitis and abscess is often difficult, if not impossible, yet having come to the conclusion that we have a localized collection of pus to deal with, there are certain symptoms which would guide us in defining its position. In cerebellar abscess we should expect severe occipital headache, distressing vomiting, giddiness, and in cases due to ear disease, absent bone conduction.

Abscess near or in communication with the tympanum is, however, more common, especially when the tuning fork test shows the auditory nerve to have escaped. It is this form of abscess which seems to me to afford the best prospects of treatment. As I have attempted to show, it is possible in many cases to distinguish between intracranial suppuration and phlebitis, but it is not often possible to distinguish positively between diffuse and localized suppuration. I cannot help thinking, however, that opening the skull even in the former may not expose the already doomed patient to additional risks. In these cases of purulent meningitis following ear disease, the intracranial inflammation is generally, if not always, already septic, because as a rule the putrid middle ear communicates with the meninges. It seems to me, moreover, that this condition may kill in one of two ways, viz., either by (1), compression, or (2), general sepsis. No doubt, by opening the cranium we diminish tension, and if tension, then pressure. Against the latter we are powerless in this locality. It seems to me, therefore, that in a case of suppurative ear disease which threatens to end fatally from intracranial complications,

and in which we have not special indications of phlebitis, we should operate—more especially if we believe that the inflammation has spread through the roof of the tympanum—the most common form be it remembered. Having arrived at this conclusion, we next ask, What is the best method of operating? It has been proposed by Sutphen[1] to force a trochar through the roof of the middle ear, but even in a case where the abscess was correctly diagnosed, this method of treatment was unsatisfactory, by the author's own showing. It must be remembered that in most of these cases the abscess is in intimate relation with the roof of the tympanum, and I am strongly of opinion that it is desirable to reach this spot with as little injury to the brain and its membranes as possible. It appears to me that this end can best be obtained by making a dissection of the auricle downwards so as to define the osseous meatus. When this has been done, an opening should be made just above and in front of it—a proceeding which exposes the dura mater within a very short distance of the roof of the tympanum, with which the great majority of cerebral abscesses are in intimate relation, without any danger of wounding the middle meningeal artery. My part of this paper does not carry me further than this, and I leave it to Mr Miller to describe two cases, in which the issues were unfortunately fatal—a termination at least in one of which (for in the other we obtained no sectio) could not possibly have been averted. In that case we were enabled to diagnose the presence of phlebitis of the lateral sinus during life, and it seems to us that the presence of this complication—were it diagnosed with a fair amount of certainty—would make us hesitate to operate in future. At the same time, it must not be forgotten that it has been proved beyond doubt by Griesinger and others, that sinus phlebitis may be recovered from.

Since the above was written Professor Greenfield has published a case of cerebral abscess due to ear disease, successfully operated on by Mr Caird.[2]

Most of those present who are interested in the subject have no doubt read Professor Greenfield's paper. The patient was admitted to the Infirmary in a state of commencing coma; headache was complained of, and paralysis of the third nerve developed after his admission. There was a slight amount of offensive pus in the meatus, and the tympanic membrane showed a small perforation. The pulse was not accelerated, the temperature persistently subnormal, and there was left optic neuritis.

In papers of the kind the reader usually expects to find a critical digest of those mental processes on the part of the physician which led to his diagnosis.

Now in this case, as recorded by Prof. Greenfield, it is difficult to see on what facts he based his opinions. We are told certainly

[1] *Archives of Otology*, vol. xiii. p. 28.
[2] *British Medical Journal*, 12th Feb. 1887.

that he arrived at the view that the case was one either of abscess or of tumour, "with," to use his own words, "a very strong preponderance in favour of abscess," but the author adds, "I need not at the moment discuss the grounds of this view." There were, however, other possibilities which we might reasonably expect to find taken into account, such as sinus phlebitis and diffuse meningitis, which we have seen to be comparatively common complications of suppurative ear disease; and, moreover, it is the reason for the diagnosis of abscess which we should have expected to be put prominently forward, instead of being entirely omitted. Further on in the paper, without, however, having expressed any definite grounds for his opinion, the following sentence occurs, "There now appeared to me to be every probability in favour of the diagnosis of abscess; and for reasons which I shall afterwards discuss, it appeared to me that the evidence was in favour of the abscess being situated in the left temporo-sphenoidal lobe, and that if so, it had extended forwards and inwards, so as to occupy a position near the front and towards the inner aspect of the lobe."

I have carefully perused and reperused the subsequent sentences, and have been entirely unable to find any grounds for the supposition that the abscess extended forwards so far as to justify the seat of operation selected in this case. That it extended inwards was shown by the involvement of the third nerve, and I cannot help thinking also by the unilateral optic neuritis, which I take it may have been in part due to pressure on the cavernous sinus. That pus was reached in this case by an operation which pierced the skull so far forwards and so high up as the junction of the temporal, parietal, and sphenoid bones was, it seems to me, due to the circumstance—fortunate alike for physician and patient—that the cerebral abscess was an exceptionally large one. Had it contained one instead of two ounces of pus, I doubt whether it could have been evacuated by the opening made.

But, although Prof. Greenfield has not called attention to them, there were facts in the history of the case which pointed strongly to cerebral abscess communicating with or near the roof of the tympanum. Thus the presence of optic neuritis showed that the patient was suffering from intracranial mischief; the existence of chronic middle ear suppuration rendered it probable that he had either diffuse suppurative meningitis, phlebitis of the lateral or other sinuses, or abscess. Meningitis or phlebitis would, however, probably have caused acceleration of the pulse, elevation of temperature, and other symptoms which were not present; therefore, judging from the persistently subnormal temperature and slow pulse, the diagnosis of abscess could easily be arrived at. As to its localization, the paralysis of the third nerve showed that the collection of pus was probably in front of the tentorium, and a knowledge of the fact that most intracranial abscesses in this situation are in intimate relation with the roof of the tympanum, would naturally point to the temporo-sphenoidal lobe as the part

affected. As I said, however, before, there was nothing to justify the belief that the abscess cavity extended any great distance forwards. The size of the collection of pus was exceptional, and it was to this very fact that the success of the case was due. Now, if in such cases the operation I have proposed were adopted, it would not matter whether the abscess extended backwards or forwards provided only it was, as such abscesses generally are, in contact with the roof of the tympanum.

General Conclusions.

(1.) That in a case of chronic middle ear suppuration, whether the mastoid region be tender or not, opening the mastoid antrum should not be too long delayed if deep-seated pain in the ear be complained of, and if the general symptoms point to head mischief.

(2.) That in advanced cases we should attempt to arrive at an exact diagnosis as to the nature of the intracranial mischief, with a view to operative interference.

(3.) That we can in most cases detect sinus-phlebitis from the presence of pyæmic symptoms and from local manifestations. Thus, if the lateral sinus be involved, the region of the jugular vein will often be found tender and occasionally corded; if the cavernous sinus be at fault, we should expect œdema of the retina, and around the eye, perhaps also exophthalmus (for further information on this point I must refer to special treatises).

(4.) That having excluded sinus-phlebitis the diagnosis may be said to lie between diffuse meningitis and localized abscess.

(5.) That the situation of the intracranial suppuration will probably be behind the tentorium if bone conduction be lost, *i.e.*, if the auditory nerve is involved. That, in my opinion, this is also more likely to be the case when there is marked inflammation of the mastoid (*i.e.*, its external and posterior part), than when the pain seems only to proceed from the tympanum; but I have been able to obtain no definite proof of this. The results of aural examination must, of course, be taken together with the general symptoms in localizing the brain lesion.

(6.) That the most common seat of localized abscess and starting point of suppurative meningitis is near the roof of the tympanum.

(7.) That we cannot in most cases distinguish positively between localized and diffuse suppuration; but that in cases where pulse and temperature are subnormal, the probability is in favour of localized abscess.

(8.) That the point chosen for opening the skull in otitic cerebral abscess should be just above and in front of the osseous meatus.

(9.) That such an operation may save a number of cases of localized abscess, and may possibly benefit some in which the suppuration is more diffuse.

(10.) That the propriety of exploring the cerebellar region is not so clear, and that this operation should only be undertaken when the presence of pus behind the tentorium is almost certain.

Table of Fatal Cases of Middle Ear Suppuration causing Intracranial Complications.

No.	Author and Reference.	Age.	Male.	Fem.	Part of Ear affected.	Brain Lesion.
1	Pooley, *Transactions of Amer. Otological Soc.*, 1879.	23	0	1	Tympanum and labyrinth.	Cerebellar abscess.
2	Kipp, *Z. f. O.*,[1] viii. p. 275.	23	1	0	Tympanum and mastoid.	Abscess of temporal lobe.
3	M'Bride, *Journal of Anat. and Phys.*, xiv.	...	0	1	Tympanum, mastoid, and labyrinth.	Cerebellar abscess.
4	Schwartze, *A. f. O.*, xvii. p. 111.	22	0	1	Tympanum, mastoid, and labyrinth.	Diffuse meningitis.
5	Pooley, *Z. f. O.*, ix.	30	1	0	Tympanum and mastoid.	Diffuse purulent meningitis.
6	Pooley, *Z. f. O.*, ix.	45	1	0	Tympanum and mastoid.	Abscess of middle lobe.
7	Weil, *Betz. Memorabilien*, 1881, vol. ii.	...	0	1	Tympanum and mastoid.	Cerebellar abscess bursting through tentorium.
8	Weil, *ibid.*	...	1	0	Tympanum and mastoid.	Purulent meningitis.
9	Habermann, *A. f. O.*, xviii. p. 87.	7	0	1	Tympanum and mastoid.	Thrombosis of lat. sinus.
10	Schwartze, *A. f. O.*, xviii. p. 279.	16	1	0	Tympanum and mastoid.	Diffuse basilar sup. meningitis.
11	De Rossi, *Statistics of Clinique*, 1879, 80, abst. *A. f. O.*, xix. p. 88.	32	1	0	Tympanum and mastoid. (no perforation !)	Diffuse meningitis.
12	Moore, *A. o. O.*, xi. p. 25.	50	1	0	Tympanum and mastoid.	Cerebellar abscess and thrombosis of lat. sinus.
13	Munson, *A. o. O.*, xi. p. 29.	39	0	1	Tympanum.	Abscess of middle cerebral lobe.
14	Ryerson, *Canadian Lancet*, 1881.	a child	0	0	Tympanum and mastoid.	Pus under dura mater covering tymp. roof.
15	Bürkner, *A. f. O.*, xix. p. 244.	20	1	0	Tympanum and mastoid.	Thrombosis of lat. sinus.
16	Same as last.	17	1	0	Tympanum and labyrinth.	Diffuse meningitis and small abscess in Pons.

[1] *A. f. O.—Archiv für Ohrenheilkunde. Z. f. O.—Zeitschrift für Ohrenheilkunde. A. o. O.—Archives of Otology.*

No.	Author and Reference.	Age.	Male.	Fem.	Part of Ear affected.	Brain Lesion.
17	Stacke, *A. f. O.*, vol. xix.(abstract) p. 297.	26	1	0	Tympanum and mastoid.	Abscess of dura mater over roof of tympanum and thrombosis of lateral sinus.
18	Christinneck, *A. f. O.*, vol. xx. p. 41.	1y4m	1	0	Middle ear and labyrinth.	Purulent meningitis.
19	Mathewson, *Transactions of Amer. Ot. Soc.*, July 1882.	11	0	0	Tympanum.	Cerebellar abscess and pus over roof of tympanum.
20	Merill, *ibid.*	32	1	0	Tympanum.	Collection of pus over petrous bone.
21	Von Tröltsch, *Gesammelte Beiträge*, etc., see *A. f. O.*, xx. p. 189.	49	1	0	Tympanum, mastoid, and labyrinth.	Meningitis involving cerebrum and cerebellum.
22	Von Tröltsch, *ibid.*	30	1	0	Tympanum and mastoid.	Abscess of middle cerebral lobe and thrombosis of lat. sinus.
23	Von Tröltsch, *ibid.*	2¾	1	0	Tympanum, mastoid, and labyrinth.	Diffuse purulent meningitis.
24	Stacke, *A. f. O.*, vol. xx. p. 275.	17	0	1	Tympanum and mastoid.	Purulent basilar and spinal meningitis and cerebral abscess (right temporal lobe)
25	Jacoby, *A. f. O.*, vol. xxi. p. 73.	43	0	1	Tympanum and mastoid.	Purulent meningitis.
26	Andrews, *Med. Rec.*, America, Sep. 1883.	24	1	0	Tympanum.	Abscess of the middle lobe.
27	Same.	42	1	0	Tympanum and mastoid.	Thrombosis of lateral sinus and meningitis.
28	Same.	31	0	1	Tympanum and mastoid.	Abscess of hemisphere.
29	Bezold, *A. f. O.*, vol. xxi. p. 255.	11	1	0	Tympanum, mastoid, and labyrinth.	Diffuse purulent meningitis involving cerebellum. Thrombosis of lat. sinus. (old?)
30	Jacobson, *A. f. O.*, vol. xxi. p. 305.	29	0	1	Tympanum and labyrinth.	Thrombosis of lateral sinus.
31	Schubert, *A. f. O.*, vol. xxii. p. 63.	30	0	1	Tympanum and mastoid.	Abscess of middle lobe and thrombosis of lateral sinus.

No.	Author and Reference.	Age.	Male.	Fem.	Part of Ear affected.	Brain Lesion.
32	Schubert, *A. f. O.*, vol. xxii. p. 63.	8	0	1	Tympanum and mastoid.	Thrombosis of lateral sinus.
33	Sutphen, *A. o. O.*, vol. xiii. p. 28.	43	1	0	Tympanum and mastoid.	Abscess of anterior and middle lobes.
34	Sutphen, *ibid.*	21	1	0	Tympanum and mastoid.	Thrombosis of lateral and sup. long. sinus.
35	Kiesselbach, *Zeitungsbericht der Phys. Med. Soc.*, Erlangen, July 1884.	19	1	0	Tympanum and mastoid. (Labyrinth ?)	Abscess of temporal lobe, and lepto-meningitis.
36	Hedinger, *A. o. O.*, vol. xiv. p. 79.	63	0	1	Tympanum and mastoid.	Thrombosis of lateral sinus and jugular.
37	Hedinger, *ibid.*	50	0	1	Tympanum and labyrinth.	Cerebellar abscess.
38	Stacke and Kretschmann, *A. f. O.*, vol. xxii. p. 252.	35	1	0	Tympanum and mastoid.	Cerebral abscess over roof of tympanum and thrombosis of lat. sinus and jugular.
39	Stacke and Kretschmann, *A. f. O.*, vol. xxii. p. 253.	19	1	0	Tympanum and mastoid.	Purulent meningitis on both sides of tentorium and thrombosis of lateral sinus.
40	Rotholtz, *A. o. O.*, vol. xiv. p. 133.	20	0	1	Tympanum and labyrinth.	Cerebellar abscesses, 2 communicating with each other, and purulent meningitis.
41	Hessler, *A. f. O.*, vol. xxiii. p. 111.	8	1	0	Tympanum and mastoid.	Diffuse purulent lepto-meningitis and abscess involving occip. and temporal lobes.
42	Bull, *Norsk Magazin for Lägevidenskaben.*	16	1	0	Tympanum.	Thrombosis of lateral sinus and jugular.
43	Kretschmann, *A. f. O.*, vol. xxiii. p. 222.	9	1	0	Tympanum and mastoid.	Thrombosis of lateral sinus.
44	Kretschmann, *ibid.*	6	1	0	Tympanum and mastoid.	Thrombosis of lateral sinus.

Part II., by A. G. Miller.

Report of Cases drawn up by Thomas Mackenzie, M.B., C.M., Resident-Surgeon.

Case I. — —, aged 20, student of medicine, was admitted into the Students' Ward of the Royal Infirmary on Thursday, 18th November 1886, complaining of pain in right eye, on right side of head and face, and on right side of neck.

History.—For one or two years patient was the subject of a discharge from the right ear, which he treated by occasional syringing. This discharge, so far as he knew, was not subsequent to scarlet fever or measles. During the same period he used to salivate to excess.

About the beginning of November the discharge from the ear ceased, and patient felt perfectly well. On Wednesday, 10th November, he did his work as a dresser in Ward XVIII., and remained in Hospital till midnight to see whether any cases should be brought in. On the following morning he felt out of sorts and remained indoors. In the afternoon he had a slight rigor, followed by pain in the right ear and down the side of the neck. Dr Stewart of Merchiston attended him from this time till his admission into the Infirmary. A small blister had been applied over the sterno-mastoid to relieve the pain in the neck, and sulphate of quinine, 6 grains, had been given every four hours. Subsequently pain occurred also in the right temporal region and eye, accompanied by lachrymation, photophobia, and injection of the conjunctiva. On Sunday, 14th November, he had a severe rigor; the pain continued, but the temperature did not exceed 100° F. until Thursday morning, 18th November, when he was found to have a temperature of 103°. He was then seen by Mr Miller in consultation, and his removal to the Infirmary agreed upon.

On his admission he was seen by Mr Miller, Dr M'Bride, and Mr Berry, and was found to be suffering from intense pain in the right temporal region and on the right side of the neck. There was twitching of the muscles of the same side.

The membrana tympani was perforated, and there was a slight amount of very offensive discharge from the ear. Pupils were equal; there was some degree of photophobia; the fundus was normal. Temperature, 103°; pulse, 80. Diagnosis—Septic intracranial inflammation. The ear was syringed out with warm boracic lotion, and three leeches were applied behind the ear. The administration of quin. sulph., 6 grains every four hours, was continued; and potass. bromid., 20 grains, was given at bedtime. The leeches afforded him great relief.

19*th Nov.*—Pain still present, though not so severe. Leeches repeated. 9.30 P.M.—Slight rigor. 11.45 P.M.—Temperature, 104°·8.

20*th Nov.*, 12.45 A.M.—Restless; potass. bromid., 20 grains. 1.20.—Temperature, 102°·4. Slept at intervals from 1.20 till 6.15, sometimes one hour, sometimes half an hour, only awake a few minutes between each sleep. Taking plenty of nourishment—milk and beef-tea. Given sod. sulph. carbol., 20 grains, every three hours. 9 A.M.—Temperature, 102°·6; rigor. 9 P.M.—Temperature, 101°·6.

21*st Nov.*—Pain continues as before. The severity of the pain in the neck has increased. The lymphatics and glands on the right side of neck and back of head being very painful and enlarged. Pain is greatly aggravated by movement. 1.15 A.M.—Temperature, 103°. 4 P.M.—Temperature, 104°. 9 P.M.—98°·8. Opening through membrana tympani enlarged; ear syringed with sod. bicarb.

22*nd Nov.*—Induration on right side of neck made out to be not in sterno-mastoid, and not in veins. 1.20 A.M.—Temperature, 104°·8. 4.30 A.M.—Temperature, 103°. 8 A.M.—Temperature, 101°·8. 11 A.M.—Temperature, 104°·6. 4 P.M.—Temperature, 102°·6. 5 P.M.—Temperature, 105°. 8 P.M.—Temperature, 100°·6. 11.15 P.M.—Temperature, 101°·6. Very restless; tinct. hyoscy., 30 minims; slept well at intervals.

23*rd Nov.*—Slept soundly till 1.15 A.M., when he woke up shivering; rigor lasted ten minutes. Temperature, 105° immediately after rigor; awake until 1.30; slept well until 3.15. 4 A.M.—Temperature, 102°·6. Very thirsty since rigor at 1.5. 6.30 A.M.—Perspiring freely. During the night a considerable amount of very offensive matter was discharged from the ear. Syringing with boracic continued. 8 A.M.—Temperature, 99°·4. 5.30 P.M.—Temperature, 106°·6.

24*th Nov.*—Very restless, and delirious. Pain as before. Right ear discharging freely. External strabismus of right eye, and commencing optic neuritis, made out by Dr Wyllie. Patient very restless, and complaining of pain in head and neck; given 40 minims of paraldehyde; slept well for an hour and a half.

25*th Nov.*—Patient still very restless and unconscious. 3 A.M.—Given 30 minims hyoscyamus; slept for an hour. Temperature, 103°·6. 6 A.M.—Very delirious and restless; given 40 minims paraldehyde; slept two hours. Patient had been seen daily by Dr Wyllie and Dr M'Bride, in consultation with Mr Miller.

At the first visit on the 25th the temperature was found to be 101°·2, having fallen from 105° the day before; the optic neuritis marked, the respiration of the Cheyne-Stokes character, and the patient quite unconscious. It was agreed, therefore, that an opening should be made into the cranium, in the hope that some localized abscess might be found and evacuated. The patient was so manifestly moribund, that anything which gave him a chance seemed justifiable. The general symptoms, more especially the

fluctuating temperature, the rigors, perspiration, and pain in the region of the jugular, rendered it extremely probable that the case was one of diffuse suppurative meningitis, with thrombosis of the lateral sinus.

The consent of the parents having been obtained, the operation was performed at 4 P.M. by Mr Miller, in the presence and with the assistance of Dr Wyllie and Dr M'Bride. The patient having been placed under the influence of chloroform, a curved incision was made over the right ear, about 3 inches in length, by means of which the auricle was drawn down. The temporal fascia and muscle were then incised down to the bone, and the pericranium scraped off the squamous portion of the temporal bone for a distance of nearly 1 inch in diameter. The external auditory meatus was thus also thoroughly exposed.

Having previously ascertained that a point about a quarter of an inch above and in front of the external auditory meatus, just above the commencement of the zygomatic arch, easy access could be obtained to the middle fossa of the skull, a small opening was made at this point by chipping with a small gouge and mallet. On penetrating the thin portion of bone at this point, pus was at once obtained, amounting to only a few drops, however. The opening was then enlarged to about the size of a threepenny piece, and a probe passed in between the dura mater and the petrous portion of the temporal bone; for it was found, as expected, that the point at which the opening had been made was just above the junction of the squamous and petrous portions.

The dura mater was thickened, roughened, and quite separated from the bone, which for some distance around was felt to be bare. There was no collection of pus or abscess of any kind—little more than what came away at the first opening and what adhered to the probe being obtained.

It was agreed, therefore, to puncture the dura with a hypodermic needle. This was done in several directions, but no abscess found: the absence of pus being verified by Dr Wyllie, who examined under the microscope the small amount of fluid that was obtained in the needle. The dura was then incised and a director thrust in, but no pus found. There was sharp hæmorrhage from the incision in the dura, but this was stopped by manual pressure kept up for about half an hour.

After the operation the patient became much quieter, slept almost constantly, and on waking appeared perfectly conscious. 9 P.M.—Temperature, 102°·4; pulse, 88.

26*th Nov.*—Patient sleeping frequently and quietly; on waking quite conscious, and not so deaf as formerly. About 10.30 P.M. became gradually unconscious, and died. Temperature 107°, having steadily and rapidly risen from 100°·4 at 8.30 P.M. on the 25th.

Report of Post-mortem Examination by Dr Bruce.

— —, æt. 20, Ward XXVII. Died 26th November; post-mortem, 28th November 1886.

Dr M'Bride's Diagnosis.—Chronic suppuration of middle ear. Perforation of tympanic membrane; a small quantity of granulation in tympanum (a crucial incision in tympanic membrane, done after urgent symptoms). Intracranial suppuration, probably extending through roof of tympanum. Possibly also thrombosis of cavernous and lateral sinuses (at beginning of attack fundus was normal; two days before death optic neuritis and external strabismus noted).

Post-mortem.—Rigidity slight in legs; lividity marked; abdomen green; a semilunar incision above right ear. Auricle had been dissected down, exposing squamous temporal and osseous meatus. An opening the size of a threepenny piece made by chiseling through squamous temporal a little above osseous meatus. (This done by Mr Miller 25th November, with evacuation of small quantity of pus, the dura mater being thickened and rough.)

Head.—On raising calvarium a little fœtid pus at vertex, superficial to dura mater, having apparently passed through ruptured Pacchionian bodies. Dura mater tense; small pale gelatinous clot in superior longitudinal sinus at vertex; on reflecting dura mater on right side, a thin layer of yellow fœtid pus, of creamy consistence, in arachnoid space (pia mater underneath this externally congested with capillary hæmorrhage); on reflecting left side of dura, a very small quantity of sero-purulent fluid in arachnoid space near middle line.

On right side dura raised from squamous temporal, outer half of petrous portion, and occipital bone over an irregularly quadrilateral area, about two and a half inches in diameter; over this area the outer surface of dura is covered with a dirty grayish fœtid slough. Dark clot in left lateral sinus; right lateral sinus pinkish, soft, purulent clot; none in cavernous sinus. Perforation in upper surface of petrous bone about one-eighth inch in diameter, overlying the tympanum, and within half an inch of the perforation made by Mr Miller, about one-quarter of an inch beyond the aperture, in the petrous portion the bone was so thinned as to give way on pressure (slight). Internal ear and mastoid cells free.

Case II. — —, age 19, tailor, Keswick, Caithness. Admitted to Ward XVIII. on Thursday, 8th December 1886, complaining of pain on right side of head and neck, with discharge from right ear.

History.—About ten years ago patient is said to have had scarlet fever. For nine years he has suffered from discharge from

the right ear, which has varied in quantity and occasionally been attended by pain. Two years subsequent to the onset of the discharge from the ear he had an attack of typhoid fever.

During the New Year holidays in January 1886, when at home in Caithness, patient one day became very feverish. Next day he suffered from headache and vomiting. Small red spots appeared over chest and abdomen. Feverishness continued for three weeks, during the whole of which time patient remained quite intelligent.

History of present attack.—A fortnight prior to his admission patient complained of pain, sometimes in his forehead and sometimes at the back of his ear. On Saturday, 26th Nov., he was seen by Dr. Bramwell in the medical waiting-room, and advised to come into Hospital. On Thursday, 1st December, his nose bled freely. During the week he had at least three rigors, and vomited several times. Saturday, 3rd November, he went to his brother's house. There he had rigors daily, was very thirsty, and would drink nothing but cold water. On Saturday and Monday he vomited. On Wednesday he had two rigors—one at night, the other in the morning. As a rule the rigors came on at night or about 9 A.M. Says that he has occasionally seen double.

State on admission, 8th December 1886.—Somewhat thin; listless expression; lay on his side. Pain on right side of head and neck. Tenderness on pressure, especially in right occipital region. Sight good, no strabismus, pupils equal.

Ophthalmoscopic Examination by Dr Wyllie.—Veins at fundus engorged, otherwise normal.

Examination by Dr M'Bride.—Hearing defective on right side. Watch heard only when in contact. Tuning fork placed on vertex heard most distinctly on right side. Membrana tympani thickened and perforated.

Patient is quite intelligent, answers questions slowly and after an interval. Tongue furred and dry, intense thirst. Pulse 116, regular. Heart sounds normal. Skin hot and dry. Temperature 105° F.

Progress.—Temperature fell from 105° at 3 P.M. to 98° at 1 A.M. It then rose steadily to 103° at 7 A.M., there being a rigor at 5 A.M., which lasted for fifteen minutes.

From their experience in the former case, Drs Wyllie and M'Bride and Mr Miller at once decided that there was septic intracranial suppuration, and that the only chance which the patient had of recovery was, if possible, to find and evacuate the pus.

It was of course evident from the pain down the neck, the fluctuating temperature and repeated rigors, that even this would afford the patient but a slender prospect of recovery; for those facts, together with the general condition, history, etc., made it probable that there was phlebitis of the lateral and probably other

sinuses, and that the intracranial inflammation was rather diffused than localized.

At 12 noon, on 9th December 1886, patient was operated upon in the large theatre of the Royal Infirmary. A curved incision was made over right ear, the auricle drawn down, ext. meatus defined, temporal fascia and muscle incised down to bone a little above the ext. meatus, and the temporal bone chiseled through carefully at a point about a quarter of an inch above and in front of ext. meatus.

The dura was found separated. No pus was seen. A probe passed along under the dura came readily on an opening into the tympanum, and passed in. A drainage-tube was put in and left. The dura did not bulge, and showed no tension or diseased condition beyond being separable from the bone for some distance round the aperture into the tympanum.

The wound was dressed with protective, corrosive sublimate wool, and iodoform. The ear was syringed out every three hours with, first, a solution of sod. bicarb., and thereafter with warm boracic lotion. Internally the patient was treated with sulphocarbolate of soda and sulphate of quinine.

After the operation the temperature fell gradually to 98°·6 at 1 A.M. on Saturday, 10th December. Again there was a rigor at 5 A.M., and by 7 A.M. the temperature was up to 103°·2. The subsequent course of the temperature is well seen in the two hour temperature chart.

10*th December*.—No headache; pain in neck and shoulder. Fundus oculi as before; veins congested. Slight twitching of face; twitching and irregular movements of arms and legs. Dorsal decubitus.

11*th December* 1886, 9 P.M.—Very restless; tossing about and moaning; wanting to go home; thinking he will not get better.

12*th December* 1886.—Much better; quieter; quite intelligent; less movement of arms and legs.

13*th December* 1886, 1 A.M.—Lying on right side; drowsy, yet restless; quite intelligent; well marked Cheyne-Stokes respiration. Pupils equal, moderately contracted; conjunctivæ congested. 10 A.M. — Looks better; more intelligent; answers questions readily. Feels tired, but has no pain. Respiration regular; occasional short coughs; very tenacious sputum; mucus with a few streaks of blood. Pupils equal; icteric tinge of the conjunctivæ. 12 noon.—Chest examined by Dr Wyllie—normal. 2 P.M.—Turned on his back and jerked left arm and leg so violently that nurse could not keep on the bed clothes. Thereafter lay on his side and slept quietly. 11 P.M.—Slight rigor.

14*th December* 1886, 3 A.M.—Has been sleeping quietly; now restless; twitching of right arm. 12 noon.—Ophthalmoscopic examination by Dr Wyllie:—Right eye, distinct optic neuritis (œdematous stage); left eye, congestion of veins as before. In-

telligence remains good. Hypodermic needle introduced through dura; blood corpusles, no pus. Bile in urine.

15*th December* 1886.—Spent a very restless night; still no headache. 10 A.M.—Petechiæ on face, wrist, and knees. 12 noon. —Dura incised with tenotomy knife. Probe introduced into arachnoid space. No pus. Hypodermic needle introduced into temporo-sphenoidal lobe three times without giving any pain. 1st, Straight inwards; 2nd, Inwards and forwards; 3rd, Inwards and backwards. No pus.

16*th December* 1886, 12·30 A.M.—Respiration 56, noisy. Still intelligent; not so restless as last night. Pulse 130, weak and irregular. 9 A.M.—Pain in chest and back—right side. 4 P.M.— Petechiæ spread over chest and back. Lips livid, skin dusky and moist, with perspiration on face.

17*th December* 1886, 1 A.M.—Cheyne-Stokes respiration, 56 per minute.

18*th December* 1886, 7 A.M.—Died. No post-mortem examination could be obtained.

I wish to make only a very few remarks,—first, on the two cases just read, and next, on the operation performed.

1. Both cases died of septicæmia, and therefore were not, I think, fair tests of the risk to life incurred by the operation, the patients being septicæmic from the first.

2. Both were somewhat deceptive in their symptoms, for they apparently improved now and again. For example, Case I. improved greatly after the application of leeches, and both had frequent falls of temperature.

3. The temperature charts were very remarkable, and would have been misleading had records not been taken both frequently and regularly.

(*a.*) In the case of Case I. there was a fall from 105° at 6.30 P.M. on the 24th November to 101° at 4 P.M. on the 25th, though he was decidedly getting worse. In Case II. there was a fall for twelve hours after the operation, and for twenty-four hours before death.

(*b.*) In Case II. a record taken at 9 A.M. and 9 P.M. would have shown a decided and continuous fall of temperature from 103°·6 on the 14th December to 99°·8 on the 18th, when he died, although 104° was reached frequently during that period. In Case I. a fall of temperature was always succeeded by a rigor and rapid rise of temperature.

In regard to the operation, which I performed in both cases, I ought to say that I was guided by suggestions from Dr M'Bride, and by careful study of the relation of parts in the skull. We found that, at the spot already indicated in the record of each case, namely, about a quarter of an inch above and in front of the external auditory meatus, the squamous portion of the temporal

bone is quite thin, and an aperture made there would be just above the junction of the squamous and petrous portions, and would give easy access to the point where the inflammation had penetrated through the roof of the tympanum.

As Dr M'Bride, Dr Wyllie, and I were quite certain that the inflammatory process inside the cranium had originated from the septic condition of the middle ear, we considered it the proper thing to go for the point of contact, namely, the roof of the tympanum. And the procedure above described seemed best fitted to effect this object.

From an aperture such as I made, very easy access is obtained to the floor of the middle fossa, and to the temporo-sphenoidal lobe also, for the purpose of evacuating an abscess if there is one. The operation is very simple, requiring special care only for the chiseling. It does not seem to produce any special effect on the patient in the way of shock.

By the curved incision over the auricle (which was suggested to me by Dr M'Bride) ready access is afforded, if necessary, to the mastoid region, though this was not required in the above cases, there being no mastoid disease. In similar cases I would not hesitate to perform the operation when the patient has no other chance of recovery. I think that this operation may also be performed in cases of septic meningitis following fracture of the base of the skull implicating the middle fossa.

With the concurrence of Dr M'Bride I would also make the following suggestion. When the presence of double optic neuritis, low pulse and temperature, associated with head symptoms in a case of ear disease, render the existence of a localized abscess probable, there can be no doubt as to the propriety of trephining, and with much probability of success. When, however, the patient has a high pulse and temperature, a septic absorption curve, and other signs of diffuse septic meningitis, it may still be justifiable to operate if the patient is rapidly sinking; it must, however, be remembered that observations by Griesinger, Schwartze, and others, show that sinus phlebitis may be recovered from. But in view of the rapid advances of cerebral surgery, we think that not one opening but two might be made, the one above the other, so that systematic irrigation might be carried out. We think also that a portion of the dura mater might be removed as well as bone, and thus brain pressure be relieved, and the arachnoid space washed out; for post-mortem examination has shown that in all cases of diffuse meningitis (arising from bone disease) there is septic pus in the arachnoid space, and it is not improbable that this is the source of the septic absorption which is the immediate cause of death.

Prof. Annandale said it was well known that surgeons had for long been of opinion that when symptoms of suppuration in the

mastoid cells occurred this bone should be laid open, but it was a more recent idea to deal with abscesses inside the skull. In regard to the symptoms of those intracranial abscesses, Dr M'Bride had very properly said that as yet they were scarcely in a position to decide between a pure limited abscess and a diffuse suppuration, and this made the question of operative treatment somewhat difficult. As to the implication of the sinuses, he thought they were now in a position to tell between thrombosis of the lateral and of the cavernous sinuses. In cases in which there was a limited or even a diffuse suppuration they were bound to interfere by operation when the symptoms became serious. Where there was in addition thrombosis, the case became more difficult; but if the symptoms of suppuration were well marked, he should still be inclined to operate, as the thrombosis might be recovered from. In regard to the seat of operation, he thought Dr M'Bride had made a distinct advance in advising the operation he did in connexion with the roof of the tympanum. There was no doubt that this was the situation which was perhaps the most common for intracranial abscess. The roof of the tympanum was very thin, and he believed there was frequently an actual communication between the dura mater and the tympanum through it. This probably was the explanation why suppuration was present at this particular part, and it was a reason why an operation should be done there.

Prof. Greenfield explained that his reasons for the diagnosis he had arrived at in his case were not discussed, because the editor had written urging him to cut it down, and this he had to do to secure its early publication. These reasons he had fully discussed in a clinical lecture to his class, and Dr M'Bride had given in brief some of the mental processes by which the diagnosis was arrived at. The case was one of greater difficulty than might be surmised from Dr M'Bride's statement of it. There was no history of ear disease and no discharge from the ear when the patient was admitted. The diagnosis was arrived at mainly by exclusion of other possible contingencies after the conclusion was come to that the condition was probably cerebral. Discharge did not appear from the ear till the fifth day after admission. Dr M'Bride saw the case two days after this, when the diagnosis had been made, and he did not seem to quite appreciate the difficulties there had been in arriving at it before this discharge occurred. The special points to which Dr M'Bride had referred were the symptoms of nerve-pressure and the size of the abscess. He did not point out that this grouping of symptoms was exceptional—so much so, that he (Dr G.) had not been able to find any other case in which the same grouping had occurred. There was a certain number of cases in which there was a communication between the ear disease and the abscess; but there were others in which healthy brain substance intervened, and the abscess was at a distance from the ear. This being so, the case was an exceptional one, and the abscess had extended

forwards, and had involved the dura by the side of the sella turcica, and there implicated the optic and third nerves. This part of the diagnosis was open to abundant criticism. The case was one in which one had to go on the summation of probabilities. He was greatly guided by the close parallelism of cases previously seen. Then when evidence of ear disease came, the general condition of the patient and the emaciation, together with the other symptoms, led him to the conclusion that the case was one of cerebral abscess. He fully agreed that the abscess would probably have been reached by operating in the position recommended by Dr M'Bride and Mr Miller. The shortness of the time at their disposal prevented their having the advantage of Dr M'Bride's presence at the operation, and Mr Caird was good enough to do it at half an hour's notice. Having made the diagnosis, he left it to Mr Caird to fix the seat of operation. The operation suggested by Dr M'Bride had been performed by Mr Miller for cases in which there was a different condition of affairs. He was inclined to think that surgeons should see that there was a free exit for discharge in such cases, either by incising the tympanum, or by opening the mastoid antrum and thoroughly disinfecting the sinus open by irrigation. He did not purpose discussing the diagnosis of cerebral from cerebellar abscess, but he thought the statistics brought forward by Dr M'Bride were dangerous to rely upon. In the majority of cerebellar abscesses he had seen there had been disease of the middle ear, with no evidence of disease of the labyrinth. The involvement of the labyrinth was, he thought, a sort of accident. Although he was inclined to concur in the view that the best position for operation was that selected by Mr Miller, he held that a comparison of skulls, particularly of children, showed that the point he gave would in some instances be too low.

Dr Byrom Bramwell desired, like the previous speakers, to express his admiration for Dr M'Bride's paper; it was, he thought, a truly scientific contribution to medicine, and likely to prove of real practical value. In his experience, cases of cerebral abscess the result of ear disease were rarely met with post-mortem. During the five years that he had acted as pathologist in the Newcastle Infirmary, he had not met with a single case; and during the three years that he has served in the same capacity in the Edinburgh Royal Infirmary, he had only met with two cases. The total number of post-mortem examinations during eight years' work in Newcastle and Edinburgh was at least 900. In consequence of the limited number of cases met with, it was very difficult for any single individual to draw any satisfactory conclusions from his individual experience. In private practice he had met with several cases in which cerebral abscess seemed to be present as the result of ear disease; in two of these cases, the diagnosis had been confirmed on post-mortem examination. In all four cases of cerebral

abscess, the result of ear disease, which he had examined post-mortem, the abscess was situated in the temporo-sphenoidal lobe; in one of the cases, there was in addition an abscess in the frontal lobe and also in the cerebellum; in two of the four cases, the abscess in the temporo-sphenoidal lobe communicated directly with the tympanum. In two cases there was, in addition to the cerebral abscess, recent diffused purulent meningitis. (He had also met with one case of diffuse purulent meningitis in the Edinburgh Royal Infirmary, in which there was disease of the middle ear and perforation of the membrana tympani. Whether the meningitis in that case resulted from the ear disease or not was uncertain; in that case there was no abscess in the brain or cerebellum). It seemed to him that in looking at cerebral abscess from a clinical point of view, it was advisable to draw a distinction between those cases, on the one hand, in which there was an encapsuled abscess, and those cases, on the other, in which the abscess was diffused, or in which an encapsuled abscess was complicated with cerebritis, diffuse meningitis, or sinus phlebitis. The symptoms were quite different in the two groups of cases. An encapsuled abscess, which was not associated with cerebritis or diffuse meningitis, might be regarded as an intracranial tumour. Cases were infinitely rare in which an encapsuled abscess was present without ear disease or nose disease, or other obvious disease of the cranial bones. In most cases of this description, it would probably be impossible, certainly extremely difficult, to make the differential diagnosis—meaning by a diagnosis a logical conclusion based upon definite facts—between an encapsuled abscess and some forms of cerebral tumour, more especially syphilitic tumours in regions which were not motor. When, however, there was ear disease—say a perforation of the membrana tympani—or other disease of the cranial bones, then the differential diagnosis between cerebral tumour and encapsuled abscess became relatively easy. It was, of course, theoretically possible to have an otorrhœa and a cerebral tumour existing in the same case; but when an otorrhœa —an obvious source of cerebral abscess—was present, the probability in favour of an abscess rather than tumour became enormously magnified. It was, however, often very difficult when cerebral symptoms were associated with otorrhœa to determine whether the symptoms were indicative of the presence of a cerebral abscess or not. In one of his cases the symptoms were those of hysteria, and the case was thought to be merely hysteria with ear disease. That hysterical symptoms should be prominent in some cases of cerebral abscess was quite what we might expect, for, as Dr A. Hughes Bennett had shown, and as he himself had in some cases seen, hysterical symptoms were sometimes the only or the most prominent symptoms in cases of cerebral tumour. Pyæmia and lead poisoning were conditions which also had to be taken into account, and which should be excluded before a diagnosis of cerebral

abscess or tumour was arrived at. Another difficulty in diagnosis was due to the fact that well-marked cerebral symptoms might result from reflex peripheral irritation in ear disease. He had himself seen headache, vomiting, and convulsions produced in this way. The condition of the optic discs was, he thought, of great importance in distinguishing these two groups of cases; for if optic neuritis was present, the cerebral symptoms were obviously due to intra-cranial changes, and not merely to the peripheral irritation of ear disease. He could not, however, agree with Dr Miller, that the presence of optic neuritis in cases of ear disease with cerebral symptoms necessarily showed that the case would run on to a fatal termination, for he had himself recorded a case in which headache, vomiting, double optic neuritis, with a marked fall in pulse and temperature—symptoms highly suggestive of a cerebral abscess—were associated with, and had without doubt resulted from ear disease (with otorrhœa), and in which, under large doses of iodide of potassium, complete cure had resulted.[1] This was, however, quite an exceptional case, and he was inclined to agree with Dr Miller that the presence of optic neuritis in cases of this description was strongly in favour of operative procedure when there were localizing symptoms. Another point which Dr Miller had referred to was the effect which arrest of the ear discharge was likely to have upon the cerebral symptoms in those cases in which the otorrhœa was associated with cerebral abscess. In one of his cases, a girl died at the age of 18 as the result of the bursting of an abscess of the temporo-sphenoidal lobe, and the production of diffuse purulent meningitis. The otorrhœa, with which the abscess was associated, dated from an attack of scarlet fever in childhood; the abscess was a very large one, and its walls were so dense and thick, that he himself and others (the specimen was shown to the Northumberland and Durham Medical Society) were of opinion that it must have existed for a very long time. In that case it was noted on several occasions that when the ear discharge temporarily ceased, the patient suffered from headache and vomiting. In that case a direct communication existed between the cavity of the tympanum and the cavity of the abscess; and it seemed reasonable to suppose that the arrest of the discharge from the ear and the consequent increased tension in the sac of the abscess were the cause of the cerebral symptoms, which disappeared when the discharge again got vent. Another interesting symptom in this case was the fact that the patient could not remember two things at the same time; on several occasions she was sent to do messages in the town, and her friends remarked that if she was told to do two things, she forgot to do one of them. He would not go into the symptomatology of those cases in which a cerebral abscess was complicated with cerebritis, diffuse meningitis, or sinus phlebitis; Dr M'Bride had in his paper considered the differential

[1] *Edinburgh Medical Journal*, August 1879, page 144.

diagnosis of these conditions, and had also detailed the chief points of distinction between cerebral and cerebellar abscess. Nor would he refer to the question of the site for operation? He thought that if the observations of Dr M'Bride—that the skull sounds were absent in cerebellar abscess, but present in cerebral abscess—was proved by subsequent observations to be a fact, it would be a valuable help to differential diagnosis. He would also suggest that in cases of suspected abscess of the left temporo-sphenoidal lobe, "word-deafness" should be looked for; a lesion in this part of the brain might be expected to give rise to this symptom, which, if present, would be of importance, both as indicating the presence of an abscess as distinct from meningitis, and as a guide to the position of the abscess, and therefore to the place for operation.

Mr Caird, in reference to the seat of operation, said the case was presented to him as follows:—A patient supposed to be suffering from abscess in the temporo-sphenoidal lobe, the indications being in favour of the anterior part of the lobe being affected, and there was further a discharge from the ear. There was, however, no evidence that there was any direct communication between the diseased ear and the abscess. It might be of the nature of so-called sympathetic abscess, or of an embolic character. To get at the part where the abscess lay the seat chosen was the most convenient. The one vessel in danger was the posterior branch of the meningeal, and even if it were injured, it was completely under control. In a case of psoas abscess, even with lumbar curvature, we did not always make a posterior opening; but if there was no fluctuation or fulness behind, we opened in front where the abscess pointed. So also in the present case. There was much to be said in favour of the surgical principle enunciated in making the posterior opening, namely, at the seat of the disease; and the fact remained that lotion injected at the ear escaped from the trephine wound after operating. Still the condition of the bulging dura, the ready access obtained to the pus, and the satisfactory progress of the case, fully justified perforation at the spot selected.

Prof. Chiene said the optic neuritis and paralysis of the third nerve had not been emphasized sufficiently as suggesting in Prof. Greenfield's case that the temporo-sphenoidal lobe was affected. He had advised the incision adopted on the ground that the pus would gravitate forwards and downwards.

Dr Hughes Bennett, in response to a call from the President, said that the subject of cerebral abscess associated with ear disease was one of which he had little experience, as such cases in London seemed almost entirely to gravitate into the hands of the aurist. The treatment, however, adopted in the paper brought before the Society this evening illustrated the improved methods of dealing with intracranial disease, which had so greatly devel-

oped during quite recent times. On the general question of cerebral surgery, the first point of interest and practical importance was the fact which recent observation and experience had demonstrated, namely, that we must abandon our former "noli me tangere" notions concerning the brain. It had now been abundantly proved, first, by direct experiment on animals, and later by operation on the human subject, that this organ might be manipulated, and portions of its substance removed without any necessary danger to life. This fact having once been established, the idea suggested itself that it might be utilized for the removal of disease, which anticipation has since been amply fulfilled. Dr Bennett had, during the last year or two, witnessed many such operations, and the only one of these attended with a fatal result was the first, which was under his own care, and this, he believed, was due entirely to the very precautions which were then taken to secure safety, which precautions subsequent experience showed to be not only unnecessary, but actually injurious. He had seen cases where tumours as large as a hen's egg had been removed from the cerebral substance, requiring considerable manipulation of the brain, and leaving a cavity of great size. Yet those patients recovered their healths, preserved their intellects, and possessed fairly useful limbs. He believed that hitherto surgeons had been too timid in undertaking operations upon the brain, which, he considered, under suitable precautions, could be carried out with much less danger than was formerly supposed. If this was more generally recognised, it was probable that relief could be afforded to many other morbid conditions other than abscess or tumour. For example, he had recently seen cases of epilepsy in which the attacks had completely and permanently ceased after trephining the skull and removing the dura mater, which he supposed was to be explained by the relief from pressure thus afforded to the cerebral substance. In the same way, surgical treatment might be utilized for the removal of blood-clots, indurations, and other morbid products. Assuming such mechanical manipulation of the brain by the surgeon to be reasonably safe, the difficulty in dealing with all these cases was the accurate diagnosis of the disease, the responsibility of which usually fell on the shoulders of the physician. The problem to be solved lay not only in the localization, but in the exact definition and limitation of the lesion. They might be tolerably certain that a special portion of the cerebral substance was the seat of disease, but it would not always be so easy accurately to define its extent and limit, or to assert how much it involved neighbouring parts. These matters were obviously of great importance when considering the question of operation. Although the recent revelations of science had done much to illuminate all these difficulties, this much remained yet to be learnt, but doubtless as their knowledge advanced they will obtain greater accuracy, both as to the localiza-

tion and limitation of disease in the brain. This, combined with improved methods of surgical treatment, should enable them in the future to afford relief to a wider and more varied extent than is now attempted.

Dr M'Bride in his reply said that cases of cure of thrombosis had been recorded by Griesinger, Schwartze, and others. These were cases in which the patient died from some other affection, and an examination showed that there had been thrombosis of the lateral sinus which had not proved fatal. With Mr Annandale's suggestion as to the opening of the mastoid antrum he concurred. Personally, he preferred to leave the tympanic membrane alone in acute cases in which the ear affection was not a complication of one of the exanthemata, until the pain had lasted for some time or the symptoms became serious. The cavity was practically aseptic, and the incision let in germs. It was very rare for acute cases to produce harm; it was the chronic form that was dangerous. He regretted that Prof. Greenfield had had to cut down his paper to such an extent as to prevent his giving reasons for the diagnosis arrived at. So far as could be made out from his remarks, his diagnosis would appear to have been a sort of intuitive result of comparison with other cases; and yet, if the speaker mistook not, Prof. Greenfield had stated that the combination of symptoms in this case was one of great rarity. The grounds for the diagnosis of these cases were of vital importance to patients suffering from such conditions. He was surprised to find that he was supposed to have held the belief that the case might be one of meningitis; he had arrived at the diagnosis of abscess almost immediately he saw the case. The ear discharge, the paralysis of the third nerve, and the optic neuritis showed that there was an intracranial lesion. The normal pulse and subnormal temperature made him conclude that it was probably an abscess, and that it was in front of the tentorium because of the pressure on the third nerve. He localized it in the temporo-sphenoidal lobe, because he knew that this was the most common seat of such abscesses; and he further ventured to suggest, that it would probably be found to communicate with the tympanum.

Prof. Greenfield said he had done all that before there appeared any ear discharge.

Dr M'Bride further remarked that he did not think that Prof. Greenfield had replied to his criticism of the seat of operation. He repeated that if the abscess had contained one instead of two ounces of pus, it would not have been reached by the opening made. Prof. Chiene told them that the pressure on the third nerve indicated that the abscess had passed forwards. He thought it rather showed that the abscess passed inwards. The question of word-deafness on which Dr Bramwell had touched was a very interesting one. He could not recall a case of otitic abscess associated with this symptom, though he believed the left temporo-

sphenoidal lobe was the part of the brain most commonly affected. Of course it must also be remembered that ordinary deafness was invariably present in those cases to some extent, and that it was sometimes very marked.

Mr Miller also replied.

Special Meeting.—March 16, 1887.

Professor Grainger Stewart, *President, in the Chair.*

I. Exhibition of Patient.

Dr Allan Jamieson exhibited a case of double comedo. The patient was a man aged 29, originally by trade a blacksmith, now a lamplighter, and otherwise in good health. When younger he suffered from acne of the face; at present it was confined to the back and shoulders. The comedones were particularly large, and a peculiarity was at once observable on inspection, that many were set with great regularity in pairs, like the spots on dice. When lateral pressure was exerted on one of these, a large plug of inspissated sebum was forced out, with a black head at either end, thus showing an anatomical connexion between the ducts of two sebaceous follicles. This curious condition, which was not pathological but physiological, was first described by Ohmann-Dumesnil, of St Louis, in the *Journal of Cutaneous Diseases* for 1886. He had recorded four cases, and believed the occurrence of such not uncommon, though hitherto overlooked. As so far no portion of skin had been excised, the anatomy of double comedo had not been worked out.

II. Original Communication.

THE DYSPNŒA OF ASTHMA AND BRONCHITIS: ITS CAUSATION, AND THE INFLUENCE OF NITRITES UPON IT.

By Thomas R. Fraser, M.D., F.R.S., F.R.C.P. Ed., Professor of Materia Medica and of Clinical Medicine in the University of Edinburgh.

Dyspnœa, or difficulty in breathing, is one of the most common of symptoms in disease, and especially in disease of the respiratory apparatus, with each of the many pathological affections of which it may be associated. My remarks, however, shall be restricted to its manifestation in asthma and bronchitis. In the former disease it presents itself as an urgent orthopnœic breathlessness, and in the

latter as a sensation of constriction or weight in the chest, and a difficulty in the performance of respiration. The breathlessness, or dyspnœa, in each of these diseases is, no doubt, dependent on defective aëration of the blood, caused either by insufficient contact of air with the bloodvessels in the pulmonary vesicles, or by insufficient movement of air in the air-passages. Insufficient contact occurs, for example, when the air vesicles are filled with liquid or semiliquid contents, and it is clearly a cause that is to some extent productive of the dyspnœa of bronchitis in many of its forms and stages, as well as of the dyspnœa of several other diseases of the lungs. Insufficient movement of air occurs when the calibre of the air-passages is reduced, or when from any cause the expansion or retraction of the chest is impeded; and this imperfect movement of air is usually recognised as a cause of the dyspnœa that occurs in both asthma and bronchitis.

Asthma.

The symptoms of asthma are, indeed, typically those that are to be associated with imperfect movement of air. They have been thus described by Riegel:[1]—"The patient having gone to bed perfectly well, is suddenly awakened in the night, while sleeping quietly, by an intense sense of oppression and anxiety. Breathing is very laborious, and the respiration is attended with audible whistling and rattling, and the dyspnœa rapidly increases to an excessive degree. The cyanosis increases from minute to minute, the face becomes bluish-red and turgid, the eyeballs protrude, the patient supports himself on both arms to struggle powerfully for air, and the face becomes bathed in perspiration. The patient can no longer get his breath in the recumbent position, and often assumes the most varied attitudes in order to appease in a measure his craving for air. . . . Soon the patient hurries to the window to struggle for a mouthful of fresh air. In spite of all this, he does not appease his craving for air, even by the forcible action of all his auxiliary muscles. The paroxysm continues at its height for a long time—one, two, or more hours—and then it gradually subsides. The respiration becomes easier again, the cyanosis disappears, the patient gradually feels freer and freer, and then drops off into a quiet, deep, uninterrupted sleep."

Now, what is the cause of this intense and distressing dyspnœa? No anatomical lesions are found which are sufficient to account for it. It is regarded as a functional disorder produced by alterations in normal physiological conditions, or by temporary structural changes which disappear with the asthmatic paroxysm.

Speculation has been active, however, in advancing hypotheses regarding the nature of the functional or temporary structural disturbances which so obviously produce an obstruction to the movement of air along the respiratory passages. Setting aside the

[1] Ziemssen's *Cyclopædia of the Practice of Medicine*, 1877, vol. iv. p. 557.

theories that were originated previously to the discoveries of Lænnec and Auenbrugger in physical diagnosis, when asthma was a term applied not only to pulmonary dyspnœa, but also to the dyspnœa of diseases of the heart and large bloodvessels, of the pleura, glottis, stomach, and other organs and parts of the body remote from the lungs, it is probable that the first certain and firm basis of knowledge as to its causation was supplied by the demonstration by Reisseissen,[1] Prochaska, and Kölliker, of the existence of a muscular structure in the bronchi. The demonstration of the contractile power of this muscular structure by Williams[2] and Longet,[3] amply confirmed by the subsequent experiments of Paul Bert[4] and others, led to a revival of the old and, for a time, discredited view, that asthma is produced by a spasmodic affection of the muscles and nerves of respiration. The asthma convulsivum of Willis then became the asthma of bronchial spasm of Cullen, Romberg, Bergsen, Trousseau, and Salter; and for more than half a century was the generally accepted doctrine, notwithstanding the enunciation of other theories, usually of a purely speculative character, such as those of Todd, Brée, Budd, Walshe, and others.

The most formidable attacks made on the theory of bronchial spasm, however, were probably those of Wintrich, in 1854, and of Weber, in 1872; as the important hypothesis of Leyden,[5] that the asthmatic paroxysm is produced by irritation of the vagus terminations in the bronchi, by minute sharp-pointed crystals, involves as an explanation of the paroxysm a reflex spasm of the bronchial muscles.

Wintrich[6] denied that spasmodic contraction of the bronchi is possible, and maintained that the only explanation consistent with the phenomena is to be found in tonic spasm of the diaphragm alone, or of the diaphragm and muscles of respiration together. He was led to adopt this theory from the results of some experiments which appeared to show that the bronchi did not contract under stimulation, and from a belief that the enlargement and hyper-resonance of the lungs, which nearly all observers had recognised during the paroxysm of asthma, could not be explained by spasm of the bronchial muscles. His opinions were supported by Bamberger,[7] who further pointed out that in a few cases of asthma the lower limit of hepatic dulness remains unchanged during both expiration and inspiration, at the line of deep inspira-

[1] *Ueber den Bau der Lungen.* Berlin, 1822.

[2] *Transactions of the British Association for the Advancement of Science*, 1840, p. 411.

[3] *Comptes rendus des sciences*, 1842, t. xv. p. 500.

[4] *Leçons sur la physiologie comparée de la respiration*, 1870, p. 379.

[5] Virchow's *Archiv*, 1872, Bd. 54, p. 324.

[6] Virchow's *Handbuch der speciellen Pathologie und Therapie*, 1854, Bd. v.; and *Krankheiten der Respirationsorgane*, Erlangen, 1855–57.

[7] *Wurzburger medicinische Zeitschrift*, 1865, Bd. vi.

tion. Wintrich's opinions and statements have not remained unchallenged. The most damaging criticisms they have sustained have been from Biermer,[1] who justly occupies the position of being one of the ablest supporters of the old theory that asthma is caused by spasm of the bronchial muscles. Biermer has the further merit of having prominently shown that asthma is characterized by expiratory dyspnœa, which distinguishes it from the dyspnœa of obstruction in the larger air-passages, where the embarrassment is more decidedly during inspiration. He endeavours to prove that spasm of the bronchi is able to cause enlargement of the thorax, increased percussion resonance over the lungs, descent and restricted movements of the diaphragm, and relative difficulty of expiration, as contrasted with inspiration; and thus he apparently succeeds in advancing a sufficient explanation of the phenomena of asthma.

The other most formidable opposition which the doctrine of bronchial spasm has encountered may, for convenience, be associated with the name of Weber, although his theory seems to be but a modification of that previously advanced by Traube. Weber[2] ascribed the asthmatic attack to a sudden congestive thickening of the bronchial mucous membrane through the agency of vasomotor nerves, and he compared the changes that were thereby produced to the local swelling and abnormal secretion of the nasal mucous membrane, which in many persons are produced by catarrh. In so far as the causation of the asthmatic dyspnœa is concerned, this theory also agrees with the old supposition that the retrocession of certain cutaneous eruptions is productive of asthma, revived in more modern times by Waldenburg,[3] in his so-called herpetic asthma, and also by Sir Andrew Clark,[4] in a paper published last year on the theory of bronchial asthma.

The three explanations of the production of the asthmatic paroxysm, which seem at the present time to be maintained more than any others, are, therefore, embodied in the theory of bronchial spasm, in the theory of spasm of the diaphragm, associated, or not associated, with spasm of the other ordinary or extraordinary muscles of respiration, and in the theory of constriction of the bronchial tubes by swellings of a hyperæmic, herpetic, or urticaria-like character.

The existence of these contending theories is a sufficient proof of the difficulties that are encountered in explaining the dyspnœa of asthma. The observations of symptoms, the assistance that has been derived from advancements in the physiology of the respiratory and nervous systems, and the great increase in knowledge

[1] *Ueber Bronchialasthma, Sammlung Klinischer Vorträge*, 1875, 12, p. 39.

[2] *Ueber Asthma Nervosum. Tageblatt des* 45, *Versammlung deutscher Naturforscher und Aerzte zu Leipsic*, 1872, p. 159.

[3] *Berliner klinische Wochenschrift*, 1873.

[4] *The International Journal of the Medical Sciences*, January 1886, vol. xci. p. 104.

of the pharmacology of the substances that are used as remedies, do not appear to have entirely solved the difficulties. No doubt the second theory, that of Wintrich, has sustained from Biermer a more damaging criticism than either of the two others has yet met with; and it may be regarded as demonstrated that spasm of the diaphragm, combined or not combined with spasm of the muscles of respiration, is not the essential or primary cause of the symptoms of asthma, however such spasm, in some cases and in some degrees, may occur as a secondary condition during the paroxysm. That able and trained observers are divided in their belief as to the correctness of the other two theories, is shown by the statement of Dr Geddings,[1] of America, that the retrocession of cutaneous eruptions as a cause of asthma, has of late years "found but few advocates among intelligent physicians;" and of Riegel,[2] that the several grades of asthma "can be explained by the mere tumefaction of the mucous membrane, seems to me improbable;" while, on the other hand, Sir Andrew Clark[3] affirms "that the bronchial spasm theory of asthma is either inadequate to explain the phenomena of the paroxysm, or is not in harmony with the present state of physiological and pathological knowledge."

It seems obvious that some additional facts are required before the truth can be arrived at. The obtaining of such facts is desirable, not merely because of the interest that is attached to the elucidation of the pathogenesis of this as of all diseases, but much more importantly, on account of the basis that would thereby be gained for the proper application of remedies. A very different treatment, for example, would be suggested for the cure of a dyspnœa dependent on stenosis of the bronchial tubes caused by hyperæmia, from the treatment of a dyspnœa dependent on stenosis caused by spasm of the bronchial muscles.

In considering the problem that is presented, we may assume that stenosis of the bronchial tubes is present. It is, indeed, impossible to overlook the significance of what are, after all, the most constant, as well as the most prominent, of the physical signs that accompany the asthmatic paroxysm. On auscultating the chest, there are heard râles of a snoring, cooing, and whistling character, unaccompanied during a part of the paroxysm, in most cases, by any moist sounds, and, in not a few cases, heard during the entire paroxysm unassociated with any moist sound, and even terminated, as Graves[4] has pointed out, without any expectoration whatever. The bronchi in which these sounds occur are furnished with blood-vessels which might dilate and produce hyperæmic swellings; they are also furnished with muscles which might contract spasmodically

[1] Pepper's *System of Medicine*, 1885, vol. iii. p. 193.
[2] Ziemssen's *Cyclopædia of the Practice of Medicine*, 1877, vol. iv. p. 554.
[3] *Loc cit.*, p. 110.
[4] *Clinical Lectures on the Practice of Medicine*, 1864, p. 507.

and here and there produce constrictions. The possibility of the latter causation of constriction cannot, I think, admit of a doubt, since the discoveries of the earlier investigators have been so amply confirmed by Paul Bert, and by Graham Brown and Roy.[1]

It occurred to me that in deciding between the two theories of the causation of the asthmatic paroxysm which seem at present to hold the field, some assistance might be derived by determining if the auscultatory phenomena to which I have referred can be modified, and simultaneously the dyspnœa reduced or removed by the action of any pharmacological agent that markedly influences the contractility of muscle, and especially of non-striped muscle. It is well known that many substances relieve the dyspnœa of asthma—such substances, for example, as atropine, morphine, and chloral—but their influence upon the auscultatory phenomena has not, so far as I know, been investigated. In the case of the substances I have mentioned investigation of this kind is not, indeed, likely to afford distinct or incisive results, as their influence on the dyspnœa is uncertain, and usually, but slowly, produced, and as they involve in their sphere of action many parts of the nervous system; while it has not been proved that independently of this involvement they influence the contractility of non-striped muscle in a very distinct or powerful manner.

In the absence of evidence of the existence of any substance that rapidly and distinctly modifies the contractility of the bronchial tubes, the analogy in structure and nerve relationship between the bloodvessels and the bronchial tubes, suggested that the most appropriate substances to be employed for the purpose I have stated would be those which are capable of modifying the contractility of bloodvessels by direct contact with them. Nitrite of amyl has been shown to possess this action, and the probabilities are in favour of its being possessed also by other nitrites and by substances that have essentially the same pharmacological action.

It seemed advisable to ascertain positively, in the first place, if all the chief nitrites possess this action, and if so, to what extent they severally exert it. I was fortunate in inducing Mr Sillar to undertake a series of experiments having these objects in view. The experiments entailed a large amount of patient observation, and they were made with great care and with every precaution to insure accuracy. The mode of procedure was as follows:—The brain and spinal cord having been destroyed in a frog, the heart of the animal was exposed and all the bloodvessels connected with it, except the left aorta, and the veins opening into the sinus venosus were ligatured. A canula was then tied into the left aorta and connected with a tube leading to reservoirs, placed always at the same height above the frog. The contents of any one of the reservoirs could be caused to flow into the aorta by

[1] *The Journal of Physiology*, vol. vi., 1883; Appendix, p. xxi.

opening or shutting clamps that were placed on the tubes leading from the reservoirs. The rate of flow of a saline solution through the entire vascular system of the animal was first ascertained, and then a solution of the same saline containing a given quantity of a nitrite was substituted for the simple saline solution, and its rate of flow through the bloodvessels of the animal was ascertained. By this procedure the effects of contact of any strength of a solution of nitrite upon the bloodvessels could be exactly determined; for if the rate of flow were diminished, it would be shown that the bloodvessels had been caused to contract, whereas, if the rate of flow were increased, it would be shown that the bloodvessels had been caused to dilate. The nitrites that were tested were nitrite of amyl, nitrite of ethyl, and nitrite of sodium. Without entering into details, I will content myself with stating that the general result was that each of these nitrites produced by contact a decided dilatation of the bloodvessels, in a few instances so great that the passage through them of the solution was doubled in its rate; and that dilatation occurred, usually in less than a minute after the nitrite had entered the bloodvessels, and was continued for periods varying from thirty to ninety minutes. Nitrite of sodium was found to be the least powerful, and nitrite of amyl the most powerful dilator of bloodvessels, nitrite of ethyl occupying an intermediate position. The difference is indicated by the statement that whereas a solution of 1 in 100,000 of nitrite of amyl was sufficient to cause a marked increase in the rate of flow through the bloodvessels—indicating a distinct dilatation of their walls—it was necessary to employ a solution of 1 in 10,000 of nitrite of sodium to produce a nearly equal effect.

The action of nitro-glycerine was not examined in the same manner, as the conditions probably required to effect its conversion into a nitrite, which exist in the blood of a warm-blood animal, could not be obtained in the saline solutions substituted for the blood in these experiments.[1]

A few experiments were, however, also made with alcohol and chloroform. Somewhat concentrated solutions of the former produced dilatation; but no constant results were obtained with chloroform, the evidence, on the whole, pointing to an absence of any dilatation under the contact of this substance with the bloodvessels.

It was thus shown that very dilute solutions of nitrites, apart altogether from any influence they may exert on structures at a distance, produce dilatation of the walls of bloodvessels as a result

[1] Since this sentence was written, Dr Atkinson, in the course of an elaborate research on nitrites made in my laboratory, has found, in experiments similar to those above described, that nitro-glycerine in very dilute solutions powerfully dilates the arteries and capillaries. This local action of nitrites has also been recently shown to occur in warm-blooded animals by R. Kobert (Ueber die Beeinflussung der peripheren Gefässe durch pharmakologische Agentien. *Archiv für experimentale Pathologie und Pharmakologie*, Bd. 22, 1886, p. 77).

of contact with them. If they could be shown likewise to modify in asthma the condition of the bronchial tubes, whose anatomical relations to bloodvessels are so marked, it is obvious that an important step would be gained in deciding which of the theories of the causation of the asthmatic paroxysm is the correct one. In the absence of any direct experimental method for ascertaining the state of the bronchi, and especially for estimating the changes that might be produced in them by medicinal agents, during an asthmatic paroxysm, it seemed to me that the observation of the auscultatory phenomena, which have, by nearly universal consent, been explained by stenosis of the bronchi, would be likely to supply important evidence.

My first observation was made in 1880, on a patient, Jessie L., twenty-two years of age, suffering from asthma and from excitement of the circulation and slight enlargement of the thyroid gland. She was one of three sisters who presented, in various forms, the symptoms of exophthalmic goitre. The dyspnœa had lasted for several weeks; it was most severe at night, but occasionally manifested itself during the day. When the observation was made (August 14, 1880) she was sitting up in bed suffering from great breathlessness. The pulse was 100 and the respirations 28 per minute. On auscultating the front of the chest, it was found that expiration was markedly prolonged, and that both inspiration and expiration were accompanied with cooing, whistling, and creaking râles, and with occasional medium crepitations.

At 1 55′ P.M. she began to inhale 10 minims of nitrite of amyl placed on blotting-paper at the bottom of a small glass tumbler, and she continued inhaling for about one minute and fifty seconds, the chest being continuously auscultated during the observations.

At 1 56′ 30″, the face was flushed, and the pulse was 120 per minute.

At 1 57′, the cooing, whistling, and creaking râles had entirely disappeared, and the patient spontaneously remarked that her breathing was easier, and that the sensation of tightness had disappeared from the chest.

At 1 58′, the râles had returned, but as yet to only a slight extent; the breathing had become more difficult, and the pulse was 95 per minute.

At 2 4′, the pulse was 96, and the respirations 28 per minute, while the breathing was as difficult, and the râles as loud and continuous as they had been before the inhalations.

From 2 6′ to 2 7′ she again inhaled nitrite of amyl.

At 2 6′ 30″, the face was flushed.

At 2 6′ 45″, the flushing had increased, the râles in the chest had entirely disappeared, and the patient stated that the breathing was perfectly easy.

At 2 6′ 50″, the pulse was 122, and the respirations 30 per minute, while the breath sounds were still unaccompanied with râles.

At 2 9′, cooing sounds were occasionally heard.

At 2 11′, the cooing sounds continued, but the breathing was still easy.

At 2 12′, the breathing was embarrassed, and cooing, creaking, whistling, and crepitant râles were audible, though they were not so continuous as immediately before the second inhalation.

At 2 19′, the pulse was 95, and the respirations 30, while the auscultatory phenomena and the difficulty of breathing were as pronounced as before the administration of nitrite of amyl.

From 2 22′ to 2 23′ she, a third time, inhaled nitrite of amyl.

At 2 22′ 30″, the face was very red.

At 2 22′ 50″, the pulse was 126, and the respirations 23 per minute, while the râles had entirely disappeared, and the breathing was again, in her own words, "quite easy."

At 2 25′, the pulse was 90, and the respirations were 28 per minute; the redness of the face had completely disappeared, the breathing was slightly embarrassed, and cooing and creaking râles were occasionally heard.

At 2 28′, the breathing was as much embarrassed as it originally had been; and with the return of dyspnœa there was a complete return of the auscultatory phenomena that had been present before the first inhalation of nitrite of amyl.

There had been no cough nor expectoration from the commencement to the termination of the observations. The patient stated that the breathlessness and sense of tightness in the chest had been entirely removed for a time by the inhalations, and the only unpleasant effect they seem to have produced was a briefly lasting sense of fulness in the head.

To illustrate more clearly the relationship between the effects on the asthma and on the circulation, I would refer to the pulse-tracings taken frequently during the observations (Figs. 1 to 12). They show, in a very remarkable manner, a coincidence between the fall of blood tension and the cessation of the dyspnœa and auscultatory phenomena, and also between the return to the original state of the blood tension and the reappearance of the dyspnœa and auscultatory phenomena. As the lowered blood tension is accompanied with acceleration of the heart's contractions, it can only be accounted for by the dilatation of bloodvessels.

Fig. 1.—Before first inhalation. Pulse 100, respirations 28 per minute. Breathing much embarrassed, râles abundant.

Nitrite of Amyl Inhaled during nearly Two Minutes.

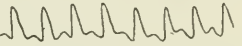


Fig. 2.—Two minutes after inhalation commenced. Pulse 120 per minute. Breathing quite easy. No râles.

Fig. 3.—One minute after inhalation ceased. Pulse 96 per minute. Breathing slightly embarrassed. Râles occasionally heard.

Fig. 4.—Seven minutes after inhalation ceased. Pulse 96, respirations 28 per minute. Breathing embarrassed. Râles nearly continuous.

Second Inhalation of Nitrite of Amyl during Two Minutes, begun Nine Minutes after the First Inhalation ceased.

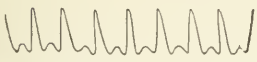


Fig. 5.—Fifty seconds after second inhalation commenced. Pulse 122, respirations 30 per minute. Breathing perfectly easy. No râles.

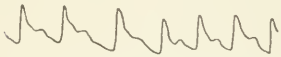


Fig. 6.—Two minutes thirty seconds after second inhalation ceased. Pulse 96, respirations 28 per minute. Breathing easy. Occasional râles.

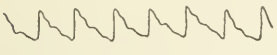


Fig. 7.—Eight minutes after second inhalation ceased. Pulse 96, respirations 30 per minute. Breathing embarrassed. Râles more frequent.

Fig. 8.—Twelve minutes after second inhalation ceased. Pulse 95, respirations 30 per minute. Breathing embarrassed. Râles abundant.

Third Inhalation of Nitrite of Amyl during One Minute, begun Fifteen Minutes Thirty Seconds after Second Inhalation ceased.

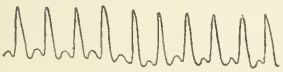


Fig. 9.—Fifty seconds after third inhalation commenced. Pulse 126, respirations 23 per minute. Breathing quite easy. No râles.

Fig. 10.—Two minutes after third inhalation ceased. Pulse 90, respirations 28 per minute. Breathing easy. Râles only rarely.

Fig. 11.—Five minutes after third inhalation ceased. Pulse 90, respiratious 28 per minute. Breathing embarrassed. Râles abundant.

Fig. 12.—Eighteen minutes after third inhalation ceased. Pulse 88, respirations 28 per minute. Râles very abundant.

Several observations were made on other patients suffering from asthmatic dyspnœa, by administering nitrite of amyl or nitrite of ethyl by inhalation. The results generally corresponded very closely with those described in the above observation.

It is apparent that, although the effects are of the greatest significance in regard to one of the main objects for which the observations had been made, they were, at the same time, of a

very transient duration. Before any further observations had been made on asthma, I had, however, succeeded in collecting a number of facts which rendered it probable that effects of a more lasting description, and therefore of greater value to therapeutics, might be obtained were the nitrites administered through the mouth or stomach. The observations in which this method of administration was followed derive an additional importance from the circumstance that they were made on patients during extremely severe dyspnœa, of a markedly orthopnœic character. These severe attacks occurred only during the night or the early hours of the morning. The occasions of their occurrence were somewhat irregular, so that it could not be anticipated with certainty that they would occur on any special night. It was, therefore, necessary to entrust the observations to those who could, at any moment, make them. They were kindly undertaken by Dr Vaughan, who was at the time acting as my resident physician at the Royal Infirmary, and by Mr Tofft, a clinical clerk in my wards, who remained in the hospital during several nights for the purpose. Both gentlemen had previously assisted me in many observations of this kind, and they were, therefore, thoroughly qualified to undertake the work.

An interesting and complete series of observations was made on a man, Hugh G., forty years of age, who had suffered from asthma for four years, and had been an inmate of the Royal Infirmary for three months. As one generally finds in cases of so long duration, emphysematous changes had been produced in the lungs, and symptoms of bronchitis were also present. The emphysema was, however, only moderate and the bronchitis slight, and frequently, for days, the symptoms of the latter were entirely absent. His sputum was usually tenacious and gelatinous, and small in quantity. Several times in each week his sleep was interrupted by severe attacks of breathlessness, which lasted from an hour and a half to three hours, and sometimes occurred twice or even thrice in one night. During the attacks the patient either sits up in bed or walks about the ward, sitting down at times to recover strength. He struggles violently for breath, inspiration and expiration succeed each other rapidly for a short time, then expiration becomes brief, the chest seems to become rigid in full inspiration, violent respiratory efforts are made with but little change in the volume of the thorax, and the extraordinary muscles of respiration are brought into play with but little result. After this state has lasted for some time, endeavours are made to cough, which are at first unsuccessful, but after a number of gasping and strained inspirations and expirations, he at last succeeds in coughing, and soon a small quantity of frothy and tenacious sputum is expectorated, when the patient either at once or soon after obtains relief. During the greater part of the paroxysm, the face and neck, and to a less extent the chest, are much congested.

Observation II.—On the 30th of December, 1886, a paroxysm began at about 4 30′ A.M. When the patient was examined, eight minutes afterwards, he was sitting up in bed holding his sides, and so breathless that he could scarcely speak. The veins of the neck were turgid, and the laboured breathing was accompanied with loud wheezing audible in the corridor of the ward at a distance of at least forty yards from the patient. The pulse was 120 per minute and feeble, and the respirations were 36 per minute.

At 4 41′, both sides of the chest were auscultated, with the result that the ordinary breath sounds were everywhere supplanted, during both inspiration and expiration, by continuous rhonchi and sibili. The time-relation of inspiration to expiration was $1:1\frac{1}{2}$.

At 4 43′, five minims of nitrite of amyl in two drachms of water were given to the patient.

At 4 43′ 30″, at the left side of the chest, the rhonchi and sibili had markedly diminished; at the right side there were no accompaniments whatever, with the exception of a few medium crepitations at the end of expiration. The face, hands, and chest were distinctly flushed.

At 4 44′, the pulse was 96, and fuller, and the respirations were 24 per minute. The patient said that his breathing was greatly relieved.

At 4 45′ 30″, the breathing at the right side was absolutely clear and vesicular; and at the left side there was only a slight sibilus during inspiration, expirations being free from accompaniments. The wheezing had by this time practically disappeared.

At 4 50′, the time-relation of inspiration to expiration was $1\frac{1}{2}$ to $2\frac{1}{2}$.

At 4 51′, there were no accompaniments whatever at any part of the chest either during inspiration or expiration, except a few medium crepitations that occurred at varying intervals and at both sides.

At 4 53′, at the right side, the breath sounds continued clear, except that now and then a distant rhonchus was heard at the beginning of expiration; at the left side, however, there was sibili throughout expiration and rhonchi during a part of inspiration.

At 4 55′, the patient stated that his breathing had become a little more difficult than it had been a short time before, and he referred the difficulty to the upper half of the sternum.

At 4 58′, the pulse was 90 and the respirations were 22 per minute, and the former was irregular in the character of the pulsations.

At 5 A.M., at the right side, there were no accompaniments excepting medium crepitations; and at the left side there was only a brief sibilus, sometimes with inspiration and at other times with expiration.

At 5 8′, the patient said he felt perfectly well.

At 5 14′, at the right side, there was a short rhonchus at the beginning of expiration, but no accompaniment whatever at the left side. The patient was now lying on the back no longer propped up. He seemed quite free from any difficulty in breathing, and he was apparently desirous to be allowed to sleep.

He was again seen at 6 10′, when he seemed to be, and expressed himself as being free from dyspnœa, but on auscultating the chest a few sibili and rhonchi were occasionally heard.

On the following day the patient was very well. His breathing was unembarrassed, but he experienced a little palpitation, and he said he had found it necessary to empty his bladder more frequently than he usually did.

Observation III.—On the same patient the following observations were made with nitrite of ethyl (nitrous ether).

On the 8th of January 1887, difficulty of breathing began to be experienced soon after 2 A.M.

At 3 15′ A.M., the patient was propped up in bed, breathing with great difficulty and showing signs of much distress. This difficulty was felt both in the act of inspiration and of expiration, but it was rather more in that of expiration, or, as the patient described it, "it was worse to get the breath out." There was also loud wheezing. A small quantity of sputum had been expectorated, which was tenacious and of a yellowish colour.

At 3 20′, on auscultation, rhonchi and sibili were heard during inspiration and expiration over both sides of the chest, but rather louder over the right than the left side. The pulse was 96 and the respirations were 22 per minute. The relation of inspiration to expiration was $\frac{3}{4} : 1\frac{1}{2}$ on both sides.

At 3 26′, ten minims of a 25 per cent. alcoholic solution of nitrite of ethyl, mixed with two drachms of water, were taken by the patient.

At 3 26′ 30″, patient said he was "not so ill."

At 3 28′, at both the right and the left sides, there were rhonchi with expiration, sibili had disappeared, and no accompaniments were heard with inspiration. The time-relation of inspiration to expiration was even on the right side, 1 : 1; and on the left, $\frac{3}{4} : 1$.

At 3 31′, the pulse was 89 and the respirations were 24 per minute.

At 3 35′, at both sides, a short rhonchus was heard with inspiration, but nothing with expiration. There was also only slight wheezing, and the patient exclaimed, "I'm almost quite easy now."

At 3 36′ 30″, both sides of the chest were entirely free from accompaniments. The time-relation of inspiration to expiration was at the right side, $1\frac{1}{4} : 1$; and at the left side, $1 : \frac{3}{4}$.

At 3 41′, the pulse was 72, the respirations were 24 per minute, and the breathing was still quite clear and free from accompaniments.

At 3 51′, the patient remarked that he was "quite easy," and had "no difficulty in the least" with his breathing; on auscultation, no accompaniments were anywhere to be heard; there was no wheezing; and the time-relation of inspiration to expiration was $1 : \frac{3}{4}$. The chest was frequently auscultated from this time until 4.50 A.M., and the breathing was always found to be soft and vesicular in character, and to be entirely free from rhonchi or sibili, while during the whole of this time the patient remained entirely free from dyspnœa.

At 4 52′, slight wheezing reappeared, the pulse was 72 and the respirations were 20 per minute; and the time-relation of inspiration to expiration was, at the right side, $1 : 1\frac{1}{2}$; and at the left side, $\frac{3}{4} : 1$.

At 4 55′, at the left side, slight sibilus was occasionally heard on inspiration, but there were no accompaniments at the right side.

At 5 A.M., the auscultatory phenomena were the same as at last note, but the wheezing was more audible, and expiration seemed slightly more prolonged. The patient coughed at this time, without expectorating, however.

At 5 11″, at the right side, there was slight sibilus at the beginning of inspiration with loud rhonchus during expiration, and at the left side, while inspiration was clear there were rhonchi with expiration. The breathing was now a little embarrassed.

At 5 18′, there were a few slight rhonchi and sibili, varying much in the time of their occurrence, at both sides of the chest. The patient at one time said the breathing "is quite easy," and at another that it was "a little difficult." The pulse was 78 and the respirations were 24 per minute. He was, however, lying in a normal position in bed.

These conditions remained unchanged until 5 25′, when the observations were stopped.

Observation IV.—An observation with nitrite of ethyl was again made on this patient, on the 24th of January 1887. On this occasion, the first symptoms of an asthmatic paroxysm began to show themselves soon after midnight.

At 12 35′ A.M. the patient was sitting up in bed, supporting himself on both elbows, and breathing with great difficulty. He stated that this difficulty was more pronounced during expiration than inspiration. There was loud wheezing, audible in the corridor of the ward, at least thirty yards from the patient's bed. A little sputum had been expectorated, consisting of dark masses of a gelatinous substance.

At 12 40′, on auscultation, it was found that at the right side there were numerous rhonchi and sibili with both inspiration and expiration; and that at the left side, sibili were almost continuous through inspiration and expiration. The time-relation of inspira-

tion to expiration at both sides was $\frac{3}{4} : 1\frac{1}{4}$. The pulse was 79, and the respirations were 30 per minute.

At 12 50′, the patient received 10 minims of a 25 per cent. alcoholic solution of nitrite of ethyl in a little water.

At 12 51′, he said, "The breathing is easier." At both sides rhonchi were heard during both inspiration and expiration, but there were no sibili. The time-relation of inspiration to expiration was at the right side, $\frac{3}{4} : \frac{3}{4}$; and at the left side, $\frac{1}{2} : \frac{3}{4}$.

At 12 54′, the wheezing had become slight. The pulse was 75 and the respirations were 24.

At 12 57′, the breath sounds were at both sides quite clear, almost vesicular in character, and entirely free from accompaniments. The time-relation of inspiration to expiration was at the right side, $1 : \frac{3}{4}$; and at the left side, $\frac{3}{4} : \frac{3}{4}$.

At 1 A.M. there was no wheezing, and the breath sounds were everywhere vesicular in character. The patient was able to lie down in a normal posture. The pulse was 72, and the respirations were 20 per minute.

At 1 6′, at the right side, an occasional slight and distant rhonchus was heard at the end of the expiration; and on the left side a similar sound at the commencement of inspiration. Slight wheezing was also audible.

At 1 11′, the conditions were the same as at 1 6′.

At 1 14′, patient said he felt "quite free" in his breathing. The pulse was 72, and the respirations were 20 per minute.

At 1 18′, the patient said the breathing was "soft as if it was oiled." There were no accompaniments on auscultation; the wheezing had quite disappeared; and the time-relation of inspiration to expiration was, on both sides, $1\frac{1}{2} : 1\frac{1}{2}$.

The patient was not again examined until 2 A.M. In the interval he had remained perfectly well, and free from any difficulty of respiration. On auscultation, the breathing was everywhere vesicular in character and without any accompaniment. The pulse was 64, and the respirations were 21 per minute. On the same day at 1 P.M., the breath sounds were also perfectly normal; there was no dyspnœa, and the time-relation of inspiration to expiration was $2 : 1\frac{1}{2}$.

Observation V.—This patient when suffering, on another occasion, from a severe paroxysm of asthma, was treated with nitrite of sodium. The paroxysm began to manifest itself at about a quarter to two in the morning of the 30th of December 1886.

At 2 54′ A.M., he was sitting up in bed in great distress, suffering from a sense of great straining in the epigastrium, and he was wheezing as loudly as before the other observations that have been described. Sputum of a very tenacious character and somewhat blood-stained was being expectorated with great difficulty. The pulse was feeble, intermittent, and extremely varying in volume.

Its rate was 84, and that of the respirations 30 per minute. The time-relation of inspiration to expiration was $1 : 2\frac{1}{4}$.

At 3 A.M., there was heard on auscultation at the right side, rhonchi throughout inspiration and expiration; and at the left side, coarse rhonchi with inspiration, and rhonchi and sibili with expiration. Sibili were apparently also being produced in the throat.

At 3 2′, ten minims of a 10 per cent. solution of nitrite of sodium, mixed with a drachm of water, were given to the patient.

At 3 3′, the right side was almost free from accompaniments, but at the left side there was heard a faint rhonchus at the end of inspiration, and an occasional faint sibilus at the end of expiration. Patient "feels a lot easier."

At 3 4′, wheezing was no longer audible, and the patient said he was "quite easy." The pulse was 84 per minute, still intermittent, but a little fuller. The respirations were 30 per minute.

At 3 6′, the time-relation of inspiration to expiration at the right side was $1 : \frac{3}{4}$; and at the left side, 1 : 1.

At 3 7′, the patient said "I feel nothing at all." The breathing was quite soft and subdued at both sides, and there were no accompaniments at all.

At 3 8′, there was slight wheezing in the throat, but the breath sounds over the lungs were perfectly normal and vesicular.

At 3 12′, the patient was talking quite comfortably, and he stated that he had "no distress whatever."

At 3 13′, a small quantity of tenacious sputum was expectorated, which "came quite easy."

At 3 15′, the breath sounds were still quite free from accompaniments, except that a few crepitations were heard at the beginning of inspiration over the right lung. The time-relation of inspiration to expiration was at the right side, $1 : \frac{3}{4}$; and at the left side, $1 : \frac{1}{2}$.

At 3 17′, the pulse was 72 per minute, and rather more intermittent than formerly, and the respirations were 26 per minute.

At 3 20′, the patient continues to "feel nothing at all."

At 3 28′, the breathing was soft on both sides without any accompaniment. The time-relation of inspiration to expiration was at both sides, 1 : 1. The pulse was 79, and the respirations were 25 per minute.

At 3 51′, the conditions were the same as at last note.

At 3 53′, the breathing was still absolutely clear and soft. The time-relation of inspiration to expiration was at the right side, $1\frac{1}{2} : \frac{1}{2}$; and at the left side, $1\frac{1}{2} : \frac{1}{2}$.

At 4 7′, the condition of respiration was the same.

At 4 26′, the breath sounds were perfectly soft and normal. The pulse was 72, and the respirations were 21 per minute. The time-relationship of inspiration to expiration was at the right side, $1\frac{1}{2} : 1$; and at the left side, $1 : \frac{3}{4}$.

The observations were now interrupted until 5 53′ A.M., when it was found that slight wheezing was again audible, and that over both lungs occasional and slight rhonchi and sibili were present. The patient stated that about five minutes previously he felt tightness of the chest and wheezing "come on all at once."

At 5 56′, there were faint sibili with both inspiration and expiration at the right side, and with inspiration alone at left side. The pulse was 78, and the respirations were 20 per minute. The time-relationship of inspiration to expiration was at the right side, 1 : 2 ; and at the left side, 1 : 1¼.

At 6 5′, the patient said "the breathing is getting tighter," and the breath is "worse to come up than go down." Over both lungs sibili were heard throughout inspiration, and the time-relation of inspiration to expiration was at both sides, 1 : 1¼.

As the asthmatic condition was obviously returning, after an absence of at least two hours and a half, it appeared of interest to determine if the return could be checked and a normal state again produced by a second administration of nitrite. The dyspnœa being as yet but slight, it seemed sufficient to administer only half the original dose of nitrite of sodium.

At 6 8′, therefore, five minims of the same 10 per cent. solution, or half a grain of nitrite of sodium, were given to the patient in a drachm of water.

In less than a minute he exclaimed, "Its away."

At 6 9′, it was found, on auscultating, that all accompaniments had vanished from both sides, the breathing having become perfectly soft. The time-relation of inspiration to expiration had also become altered, for at the right side it was 1 : 1; and at the left side, 1 : ¾, showing a diminution in the duration of expiration.

The patient was finally seen at 7 15′ A.M. He had continued quite well since he had received the second dose. There had been no wheezing, nor sense of tightness, nor any form of difficulty in respiration. On auscultating over both lungs, it was found that the breath sounds were perfectly clear and soft, although a few small crepitations occurred early in expiration at the right side. The pulse was 78, and the respirations were 24 per minute. The time-relation of inspiration to expiration was at both sides, 1 : 1½.

Observation VI.—While this patient was suffering from a severe attack of orthopnœa, an observation was made on him with nitro-glycerine. The attack began at 2 A.M., on a damp and cold night (December 28, 1886). When he was seen at 2 35′ A.M. he was sitting upright in bed, holding on to it, and breathing with extreme difficulty, the difficulty being, as he described it, during both inspiration and expiration. There was also a cough which, after great and prolonged efforts, brought up sputa, copious in amount, muco-purulent, stained with blood, and very tenacious.

It was found, on auscultation, that loud and continuous sibili occurred in both lungs during inspiration and expiration.

At 2 41′, he received two and a half minims of a one per cent. solution of nitro-glycerine diluted with a drachm and a half of water.

Almost immediately thereafter he exclaimed, "Oh! it's easier," and the wheezing had almost disappeared in a few seconds subsequently.

At 2 42′, inspiration and expiration were in both lungs very much softer, sibili had almost disappeared, slight subdued rhonchus was heard at the beginning of expiration, and there were some crepitations. The pulse was 96, and the respirations were 36 per minute.

At 2 45′, at the right side, there was slight rhonchus throughout inspiration, and at the left side short rhonchus at the beginning of inspiration. The pulse was 78, and the respirations 24 per minute.

At 2 47′, wheezing had again become audible, an attack of difficult coughing occurred, and the patient stated that the breathing was again tight, but in a few seconds afterwards he said that the tightness had disappeared. The pulse was 77, and the respirations 24 per minute.

At 2 54′, the breath sounds were absolutely clear, soft, and vesicular, and without any accompaniment.

At 3 2′, while the breath sounds at the right side were perfectly normal, at the left side there were slight distinct rhonchi at the beginning of inspiration and expiration. The patient experienced a little tightness of breathing, but only during a few seconds.

At 3 6′, at both sides, there were distant sibili throughout inspiration and expiration.

At 3 10′, another attack of violent coughing occurred, when a little sputum was expectorated. The pulse was 86, and the respirations 24 per minute.

At 3 12′, there was again marked wheezing, and rhonchi were heard during inspiration and expiration over both lungs.

The observations were continued until 5 35′, and they showed a gradually increasing development of breathlessness and of the respiratory accompaniments, but neither attained the severity and urgency which had characterized them before nitro-glycerine had been administered.

Observation VII.—The effects of nitro-glycerine were well illustrated in another patient, Thomas H., thirty-six years of age, a well-built, muscular man, a joiner by occupation, whose illness had begun about fourteen weeks before the observation was made. There was no history of hereditary predisposition to asthma, and no personal history of pulmonary disease, with the exception of a single attack of acute pneumonia which had occurred four years

previously. The patient referred the origin of the asthma to a "severe cold," following exposure to a heavy rain. A week afterwards the dyspnœa appeared, and paroxysms of asthma occurred regularly every morning at 6 o'clock, and lasted for about three hours. He also had considerable dyspnœa during the greater part of nearly every day, and especially when the weather was foggy, which prevented him from working or going about, but produced no discomfort while he was at rest beyond a sense of tightness and weight in the chest. He was free from cough and had no sputum, excepting when the dyspnœa was present, and at these times his sputum was pretty copious, watery, and frothy. No evidence was obtained of enlargement of the heart; but, although the chest was well formed, the lungs were slightly emphysematous. Expansion was good, expiration was a little prolonged, and, generally, rhonchi and sibili with medium and small crepitations were audible over the greater part of the chest. The patient also suffered from headaches, which occurred in the morning after the commencement of each paroxysm, and usually disappeared toward the afternoon.

The case was, therefore, one in which chronic bronchitis was also present, and on several occasions, after the patient's admission into the hospital, observations were made while there were no paroxysms of asthma on the influence of nitrites on the symptoms of bronchitis, to some of which I shall afterwards refer. The influence of nitrites and the conditions in which the patient was placed in the hospital, appeared to lessen the severity of the asthmatic paroxysms; they became less frequent, the time of their occurrence lost the regularity it had originally possessed, the bronchitis disappeared, and the patient was dismissed on December 26, 1886, apparently cured of both asthma and bronchitis, as he had no symptoms of either disease for ten days.

After returning to his home in Edinburgh, he remained well until the 28th of December. On that day, although he had not resumed work or undergone any exposure, severe dyspnœa appeared at 3 o'clock in the afternoon, and continued until 9 o'clock on the following morning. He, therefore, again came to the hospital, and was readmitted. He remained free from dyspnœa during the afternoon, but towards evening the breathing gradually became difficult, until at about 11 30′ P.M. the difficulty had increased to orthopnœa, and the patient was obliged to sit upright in bed, supporting himself with his extended arms.

At 1 12′ A.M., the patient was in great distress, feeling, as he said, "like to choke." He stated that the difficulty in breathing was felt chiefly during expiration. He had spat a small quantity of slightly adhesive sputum. There was loud wheezing, and when the chest was auscultated sibili were heard throughout inspiration and expiration at both sides, completely masking all other sounds. The pulse was 112, and the respirations 30 per minute. The time-relation of inspiration to expiration was 1 : 3.

At 1 20′, he received five minims of a one per cent. solution of nitro-glycerine mixed with two drachms of water. There was a perfect "storm" of accompaniments when the nitro-glycerine was given. In less than thirty seconds he said, "I feel a little easier."

At 1 21′ 30″, the wheezing was less audible, and at the left side inspiration was almost clear, but there were rhonchi with expiration.

At 1 23′, the breathing was almost clear at the right side, and there was a brief sibilus on expiration at the left side. Patient said he "feels much easier."

At 1 23′ 30″, at the right side there were sibili during expiration; and at the left side, sibili during inspiration. The pulse was 114, and the respirations were 30 per minute.

At 1 26′, headache came on.

At 1 27′, he expectorated about half an ounce of frothy and slightly tenacious sputum.

At 1 28′, at the right side both inspiration and expiration were vesicular and free from accompaniments; and at the left side inspiration was perfectly clear, but very slight rhonchus occurred with expiration. The wheezing was scarcely audible, and the patient said he was "a great deal easier."

At 1 31′, the pulse was 108 and the respirations were 30 per minute. The patient said the difficulty of expiration was "nothing to speak of."

At 1 34′, the breathing was at times perfectly free from accompaniments, and then for a few seconds sibili or rhonchi were heard on one or other side of the chest. The pulse was 108 and the respirations were 28 per minute. The time-relation of inspiration to expiration was $1 : 2\frac{1}{2}$.

At 1 38′, the chest was beginning to feel tight, and the wheezing was distinctly audible.

At 1 42′, the breathing had become "a great deal stiffer"; the difficulty, according to the patient, was in "getting out breath." Over both lungs rhonchi and sibili were heard during inspiration and expiration.

At 1 50′, the above sounds were only occasionally heard, and the patient said he "feels as free as ever he was;" and his appearance was again that of ease.

At 2 2′, 2 14′, 2 27′, and 2 44′, the auscultatory phenomena continued as at last note. At 2 14′, the pulse was 106 and the respirations were 24 per minute; and at 2 27′, they were 80 and 25 per minute respectively.

At 2 58′, the time-relation of inspiration to expiration was, at the right side, $1 : 1$; and at the left side, $1\frac{1}{2} : 2\frac{1}{2}$. The character of the breathing was soft, but now and again a brief sibilus was heard, with small crepitations at the end of expiration.

At 3 15′ and 3 57′, the conditions remained as at the last note.

At the latter time the pulse was 80 and the respirations were 29 per minute; and the time-relation of inspiration to expiration was, at left side, 1 : 2.

The patient was now left to himself, apparently free from any obvious sign of dyspnœa, but still, on being asked, confessing to a sensation of slight constriction in the chest.

When he was again seen, at 4 30′ and 5 20′ A.M., he was sound asleep and breathing quietly and without wheezing.

Observation VIII.—The last case I shall describe is one which illustrates the close relationship frequently observed between bronchitis and asthma. The patient was a man, Robert B., fifty-two years of age, presenting the ordinary symptoms of pronounced emphysema and severe bronchitis, and suffering greatly from frequent periodic attacks of dyspnœa and orthopnœa. The bronchitis was manifested by coarse and medium crepitations, continuous rhonchi and sibilations, and severe cough, accompanied with an abundant mucopurulent and frothy sputum. The asthma manifested itself in paroxysms of urgent orthopnœa, occurring during the night and almost every night, and so prolonged that the patient could not obtain sleep, except in the morning and during the day.

On the 8th of January, 1885, one of the usual paroxysms began at 9 P.M. He was seen immediately afterwards, when he was sitting up in bed in great distress, with loud wheezing respiration; and it was found that rhonchi and sibili were abundantly present.

At 9 30′ P.M., a pulse tracing was taken—the pulse being 64 and the respirations 21 per minute. (Fig. 13.)

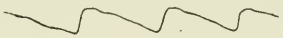


Fig. 13.—Robert B. Before nitrite of sodium. Pulse 64, respirations 21 per minute.

At 9 35′, five grains of nitrite of sodium dissolved in one drachm of water were administered.

Almost immediately afterwards, the patient felt some peculiar sensations, which he described as "the medicine going all over him and making him feel queer." In the course of a few minutes the dyspnœa was relieved, the rhonchi and sibili had entirely disappeared, and the respiratory movements were more full and more easily performed. The patient soon lay down on the bed and seemed disposed to sleep.

The pulse showed the following characters at thirty minutes, at one hour, and at one hour and a half after the nitrite had been administered. (Figs. 14, 15, and 16.)

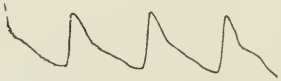


Fig. 14.—Robert B. Thirty minutes after nitrite of sodium. Pulse 76, respirations 20 per minute.

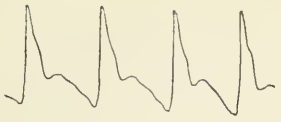


FIG. 15.—Robert B. One hour after nitrite of sodium. Pulse 76, respirations 18 per minute.

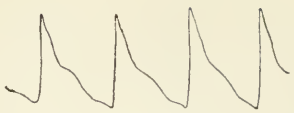


FIG. 16.—Robert B. One hour and a half after nitrite of sodium. Pulse 72, respirations 17 per minute.

The chest was frequently auscultated, and it was found to remain free from rhonchi and sibili during two hours succeeding the administration. After this time rhonchi were again heard, but only in a subdued form. The patient, however, was so greatly relieved in his breathing that he slept whenever he was left undisturbed, and he remained free from dyspnœa all night.

At two hours and at four hours after the administration, the pulse possessed the characters represented in the next two tracings. (Figs. 17 and 18.)

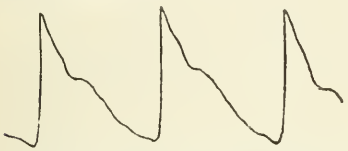


FIG. 17.—Robert B. Two hours after nitrite of sodium. Pulse 68, respirations 18 per minute.

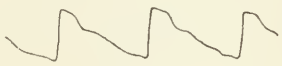


FIG. 18.—Robert B. Four hours after nitrite of sodium. Pulse 64, respirations 18 per minute.

On other occasions similar satisfactory results were obtained on this patient with nitrite of sodium and also with nitro-glycerine. The latter, however, several times produced severe headache, and, therefore, nitrite of sodium was more frequently given. While five-grain doses of it in a most marked manner subdued the severe paroxysms of dyspnœa—the patient on one occasion stating that he would have died had the medicine not relieved him—it did not cause any headache. At the same time it was found that the dyspnœa could, in this patient, generally be relieved by one or two grain doses. One administration, but only one out of a considerable number, of five grains was followed by toxic symptoms, consisting mainly of great feebleness of the circulation, which, however, quickly disappeared after the administration of a little brandy.

The administration by the stomach of nitrite of amyl, nitrite of ethyl, nitrite of sodium, and nitro-glycerine, therefore, produced the same kind of effect on asthma as that which followed the administration by inhalation of the volatile of these nitrites. A marked, and for therapeutic purposes a very important difference, was manifested in the duration of these effects, which were greatly prolonged by stomach administration.

It has been well recognised that the auscultatory phenomena,

which have been referred to, are present during the asthmatic paroxysm. It does not appear to have been distinctly appreciated that they are so intimately associated with the paroxysms, that dyspnœa is present only while they are present, and that it subsides or disappears only when they subside or disappear. Not only has this been rendered apparent by the observations I have described, but also by an observation in which the exceptional result was obtained, that a nitrite administered during a severe asthmatic paroxysm failed to produce more than an insignificant and temporary improvement in the dyspnœa, and equally failed to subdue more than to a slight extent, and for a brief period, the loud rhonchi and sibili that were present.

The observations that have been described further show that both the dyspnœa and the sounds in the chest can be made to disappear simultaneously, or nearly so, by substances whose action is to reduce, powerfully, the contractility of non-striped muscle. It appears to follow from this that the dyspnœa of asthma is caused by spasm of the bronchial muscles.

The view that this dyspnœa finds its chief explanation in spasm of the diaphragm, associated or not associated with spasm of the ordinary muscles of respiration, has, as I have already stated, received so damaging a criticism from Biermer, that its further refutation by such observations as have been brought forward seems to be almost unnecessary. The remaining view to which any importance may be attached, that, namely, of constriction of the bronchial tubes by swellings of a hyperæmic, herpetic, or urticaria-like character—whose most prominent upholders are Weber and Sir Andrew Clark—presupposes for the production of the swellings a dilated state of the bloodvessels of the bronchial tubes. The means which I have successfully employed, however, for controlling and checking the asthmatic paroxysm are the very means which should, according to this theory, be the most efficient that could have been selected for increasing the paroxysm and rendering it more prolonged. There is no fact in pharmacology more certain and undoubted than that nitrites produce rapid and great dilatation of the bloodvessels throughout the body.[1] In the first observation I brought forward, and I have others of a like kind, this dilatation was produced at the moment when the dyspnœa disappeared; it was maintained while the dyspnœa was absent; and it gave place to a normal condition of the bloodvessels when the dyspnœa returned. It seems, therefore, to have been abundantly shown that the theory of the production of

[1] Although it has not been proved by direct observation that nitrites dilate the bloodvessels on the surface of the bronchi, there is no reason to doubt that they do so; while the fact that these bloodvessels are derived from the aorta and intercostal arteries, and that they possess the same structure as the other bloodvessels of the systemic circulation, afford a strong presumption in favour of their being dilated by nitrites in common with the other bloodvessels of this system.

asthmatic dyspnœa, by swellings of the bronchial mucous membrane of a hyperæmic or inflammatory kind, can no longer be maintained.

The conceptions of the conditions that immediately produce the asthmatic dyspnœa or orthopnœa have been obscured by the numerous and unharmonizing theories that have been propounded. If the results of the observations I have brought forward should produce the impression upon others which they have produced upon me, I believe these obscurities will to a great extent disappear, and the old doctrine, that the asthmatic paroxysm depends immediately upon spasm of the bronchial muscles, will be more firmly established in the position which it had formerly occupied. At the same time, it is not to be supposed that this doctrine is incompatible with the view that, in a secondary manner, and as a result probably of the dyspnœa which has already been caused by contractions of the bronchial muscles, spasmodic contractions may also be originated in the diaphragm and in other of the ordinary muscles of respiration.

The success in any disease of a therapeutic agent, whose action is a known one, affords valuable evidence of the correctness of the theory of the causation of that disease. Such evidence appears to be afforded in a very incisive manner by the influence of nitrites in asthma. In this disease many other remedies have also been found to produce benefit. Probably this benefit has been more markedly associated with the inhalation of the smoke of certain solanaceous plants, of nitre, and of several patent medicines in the form of powders, than with any other remedies. The cause of the benefit which these substances produce is, however, almost unknown, and such speculations have been advanced as those of Oertel in his *Treatise on Respiratory Therapeutics*,[1] that the fumes of stramonium and of nitre are beneficial because they stimulate the air passages, giving rise to violent coughing and copious expectoration, and not because they act anæsthetically and antispasmodically on the bronchioles and lungs. Their influence upon the essential phenomena of asthma, and especially upon those phenomena that imply spasm of the bronchial tubes has not, indeed, been investigated, nor is there much knowledge as to the composition of the patent asthma remedies. The extensive use of these remedies suggests that advantage must be gained from their employment; and it is a common experience to meet with patients who have a greater faith in their power to give relief, than in the arsenic, or iodide of potassium, or lobelia which may be recommended to them by their medical advisers. So much have I been impressed with this circumstance, that I have procured several of these patent medicines for the purpose of having their composition, and the composition of the products of their combustion, determined.

[1] Von Ziemssen's *Handbook of General Therapeutics*. Translated by J. Burney Yeo, M.D., 1885, p. 178.

This has been done for me by my assistant, Dr Atkinson, with the results noted in the subjoined table:—

	Before burning, infusion made with water contains			After burning, smoke contains	
	Nitrite.	Nitrate.	Pupil dilator.	Nitrite.	Pupil dilator.
1. Maokill's Asthma Cure (Hamilton)	None	Distinctly	Distinctly	Distinctly	Distinctly
2. Himrod's Asthma Cure (Himrod Manufacturing Co.)	„	Abundantly	„	Abundantly	„
3. Hinksman (Carluke)	„	Distinctly	„	Distinctly	„
4. Seuler's Asthma Remedy (London and Milwaukee)	„	Trace	„	None	„
5. Green Mountain Asthma Cure	„	„	„	„	„
6. Binning's Asthma Cure	„	Distinctly	„	Distinctly	„
7. Girdwood's Patent Asthma Cure (Belfast)	„	„	„	„	„
8. Edward's "Valley Moss" Asthma Cure	„	Trace	„	None	„
9. Ozone Paper (Huggins, London)	„	Abundantly	None	Abundantly	None
10. Hockin's Remedy for Asthma and Bronchitis (Ryde, Isle of Wight)	„	None	Distinctly	None	Distinctly
11. Papier Fruneau contre l'Asthme (Fruneau, Nantes)	„	Abundantly	None	Abundantly	None
12. Dr Palmer's Antiasthmatic Papers (Simpson & Co., Dublin)	„	„	„	„	„
13. Joyes' Cigares Antiasmatiques	„	Trace	Distinctly	None	Distinctly
14 Argo Cigarettes (Blair, Perth)	„	Faint Trace	„	„	„
15. Cigarettes Indiennes (Grimault & Cie, Paris)	„	None	Trace	„	Faint trace
16. Kay's Stramonium Cigarettes (Stockport)	„	„	Distinctly	„	Distinctly
17. Dr Douglas's Maori Cigarettes (Perth)	„	Trace	„	„	„
18. Marshall's Cubeb Cigarettes (Horner, New York)	„	None	Trace	„	Faint trace

Arsenic was searched for in all of the above preparations, but was not discovered in any of them. Nitrites were not found in the smoke of any of the cigarettes examined; but of the other preparations, in 66 per cent. the products of combustion contained nitrites, and in large quantity in 50 per cent. of them.

While these results supply a sort of confirmation to the value of nitrites in asthma, which the observations that have been described so strongly suggest, it is undoubtedly the case that the best therapeutic effects are not obtained by the inhalation of nitrites, but by their administration through the stomach. The facts that have been stated seem to justify the assertion that their administration in this manner in asthmatic dyspnœa or orthopnœa is entitled to rank as one of the most valuable of the applications of pharmacology to the treatment of disease, an application at least as valuable as that in the painful angina of aortic disease, to which nitrites are at present almost restricted.

Bronchitis.

Having ascertained that the dyspnœa of asthma and the auscultatory phenomena which accompany it are produced by spasm of the bronchial tubes, I next applied the same method of investi-

gation to the dyspnœa of bronchitis, where also dry râles, having the same characters as those that occur in asthma, are met with. In bronchitis, the dyspnœa is undoubtedly a symptom which is less urgent than in asthma, but still it is often so prominent as to add greatly to the distress of the patient. Its causation is admittedly a more complex one in bronchitis than it is in asthma. In the former disease, it is usually associated with physical signs of a more varied description; for it may be accompanied not only with dry râles, but also with many varieties of moist sounds. The explanation of the production of the latter is not, so far as I know, a matter of doubt or ambiguity, but some difference of opinion exists as to the explanation of the former.

The impression in my own mind has until lately been that the dry râles, the rhonchi and sibili, the snoring, cooing, and whistling sounds, are produced by swelling or engorgement of the mucous lining of the bronchial tubes, or by constrictions of these tubes caused by deposits of adhesive mucus or other products of inflammation; and that these sounds, because they were indications of these or similar changes in the bronchial tubes, were among the most important of the symptoms of bronchial inflammation.

A spasm element, whose influence upon the physical phenomena of bronchitis was, however, by no means easily definable, entered into the conception of the disease in those cases where dyspnœa was specially urgent, or where it interrupted the ordinary course of an otherwise continuous slight dyspnœa by periodically assuming exacerbations in the intensity of its manifestations.

A reference to the literature of bronchitis has, on the whole, confirmed the impression that what I have stated is the prevailing opinion, and the prevailing teaching on the subject. For example, the dry sounds of bronchitis, the rhonchi and sibili, are stated by Laennec,[1] Guttmann,[2] Davis,[3] Latham,[4] Hilton, Fagge,[5] Riegel,[6] Jaccoud,[7] and others, to be produced by contractions of the bronchial tubes, caused by tenacious mucus, tumefaction, engorgement, or puckerings of the mucous lining. Only a few writers, such as

[1] *A Treatise on Mediate Auscultation.* Edited by Theophilus Herbert, M.D., pp. 52, 53, 61, 64, 73, 74, 78. London, 1846.

[2] *Handbook of Physical Diagnosis.* Translated for the New Sydenham Society by Alexander Napier, M.D. 1879, pp. 159, 160.

[3] Pepper's *System of Medicine*, 1885, vol. iii. pp. 171, 178.

[4] *Collected Works.* Edited for the New Sydenham Society by Dr R. Martin. 1878, vol. ii. pp. 112, 113, 116, 117, 120.

[5] *The Principles and Practice of Medicine*, 1886, vol. i. pp. 863, 864.

[6] Ziemssen's *Cyclopædia of the Practice of Medicine*, 1877, vol. iv. pp. 354, 388, 427.

[7] *Traité de pathologie interne*, 1883, t. ii. pp. 378, 381, 382.

Niemeyer,[1] Stokes,[2] Roberts,[3] and Carmichael,[4] state that it is occasionally indicated by these sounds that the bronchi are being constricted by spasm of their muscles.

The conceptions generally prevalent on the subject may perhaps be best illustrated by the following quotations:—

Riegel, in his elaborate dissertation on bronchial catarrh, contributed to Ziemssen's *Cyclopædia of the Practice of Medicine,* states:

"The accurate determination of the character of the râles [in bronchial catarrh] is of especial importance, because we can determine thereby the special sort of alteration existing in the bronchial tubes. Thus the long-used distinction between moist and dry râles has an important significance. The former are due to the movement of thin liquid products in the trachea and bronchi, and the latter are due to the friction of the current of air against the swollen mucous membrane of the bronchi, and to the presence of very viscid products. Dry râles indicate, therefore, more or less considerable swelling of the mucous membrane, and eventually the presence of small quantities of very tenacious fluid in the bronchi." [5]

In another place, having referred to the effects of secretion in the air tubes, he proceeds to state:

"In other cases, only dry, whistling, and sonorous râles are heard, occasioned by severe swelling of the mucous membrane, and the presence of tenacious, scanty secretion." [6]

Bristowe, in his *Treatise on the Theory and Practice of Medicine,* thus explains these sounds:[7]

"The cause of rhonchus is not the bursting of bubbles or the passage of air through fluid, but the passage of air through a tube narrowed at some point either by thickening of its parietes or by the adhesion of a plug of tenacious mucus." . . . . "The pitch of the musical note depends on various complex conditions, the exact influence of each one of which it would be difficult to estimate, but is determined in very considerable degree by the size of the bronchial tube in which it is developed. Thus, as a general rule, hissing and whistling sounds, or sibilant rhonchi, arise in the smaller tubes, and grave tones or sonorous rhonchi are the product of the larger ones."

Many quotations to the same effect could be extracted from the writings of other authors.

Putting aside as a cause of dyspnœa the moist sounds, which in bronchitis imply, according to their abundance, either obstruction to the movement of air in the bronchi, or obstruction to the contact of air with the bloodvessels in the air cells, there remain for consideration the dry sounds of the different qualities of rhonchi and sibili, which share with the moist sounds a peculiar diagnostic importance in bronchitis.

[1] *A Text-book of Practical Medicine.* Translated by Geo. H. Humphreys, M.D. 1870, vol. i. p. 82.

[2] *A Treatise on the Diagnosis and Treatment of Diseases of the Chest.* Edited for the New Sydenham Society by Alfred Hudson, M.D. 1882, p. 64.

[3] Reynold's *System of Medicine,* 1871, vol. iii. pp. 891, 896.

[4] *Edinburgh Medical Journal,* Oct. and Nov. 1886.

[5] *Loc. cit.,* p. 354.

[6] *Loc. cit.,* p. 388.

[7] Third edition, 1884, pp. 386, 387.

A number of observations were made with the object of determining to what extent these sounds are modified by nitrites, and to what extent any modification produced was associated with a change in the severity of the dyspnœa that was present.

The observations were made in many forms and stages of bronchitis, but for the purpose I have in view they may be arranged in accordance with the characters of the expectoration, as, for instance, if that were mucopurulent, or serous, or glairy and adhesive, or abundant or scanty. The effects of nitrites on the dyspnœa and auscultatory phenomena, where such like variations in the expectoration existed, may be illustrated by a brief description of a few observations selected from many others that were made. In this selection, observations have been taken which will also serve to illustrate the effects of each of the nitrites administered, and of nitro-glycerine.

Observation IX.—Daniel M'D., a carter, aged 44, was admitted into the Royal Infirmary, complaining of cough, profuse expectoration, constant difficulty in breathing and general weakness. He had been troubled with cough for twenty years, but had otherwise been healthy. For the last two years he had not been able to work. His chest was somewhat barrel-shaped, and there was a hyper-resonant note over the whole of the anterior surface. The auscultatory phenomena were those of bronchitis, viz., prolonged expiration, numerous median crepitations, and an abundance of rhonchi and sibili. The heart was dilated slightly, but there was no evidence of disease of the valves.

On the 6th of October 1886, immediately before nitro-glycerine was administered, the following conditions were present: The breathing was "a little difficult," the chest feeling "very stiff." Since early morning he had expectorated a large quantity of mucopurulent sputum. At the right side there was snoring rhonchus with crepitations during inspiration, and brief rhonchus during expiration; and at the left side there was sibilus during inspiration and expiration, expiration being considerably prolonged at both sides. The pulse was 100, and the respirations were 25 per minute. At 11 35′ A.M. there were rhonchi throughout inspiration and expiration at both sides, with a few crepitations which occurred occasionally.

Fig. 19.—Before nitro-glycerine; pulse 100, respirations 26.

At 11 36′, four minims of a 1 per cent. solution of nitro-glycerine were administered in a little water.

At 11 36′ 30″, at both sides the rhonchi and sibili had entirely

disappeared, a few crepitations only remaining; and the patient said he "felt much relieved."

At 11 40′, the pulse was 98, and the respirations were 30 per minute.

Fig. 20.—Four minutes after nitro-glycerine; pulse 98, respirations 30.

At 11 41′, at the right side the breathing was quite soft, and at the left side it had the same character, but a faint distant rhonchus was now and then heard.

At 11 45′, the pulse was 102, and the respirations were 30 per minute.

Fig. 21.—Nine minutes after nitro-glycerine; pulse 102, respirations 30.

At 11 48′, the breathing was perfectly soft, and free from all accompaniments at both sides. The patient stated that he has no feeling of tightness, and that his breathing is "quite easy."

From 11 51′ to 12 38′ the chest was almost continuously auscultated, and during the whole of this time the breathing continued to be soft and vesicular, and free from rhonchi and sibili; the only accompaniments being small and medium crepitations, which were usually, but not invariably, present. During the whole of this time, also, the breathing of the patient remained altogether easy and unembarrassed. The pulse and respirations were usually slightly less frequent than they had been before the administration of the nitro-glycerine.

Fig. 22.—Eighteen minutes after nitro-glycerine; pulse 99, respirations 27.

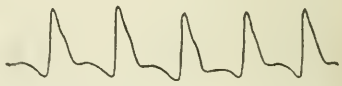


Fig. 23.—Thirty-four minutes after nitro-glycerine; pulse 101, respirations 27.

Fig. 24.—One hour and two minutes after nitro-glycerine; pulse 96, respirations 27.

The uniformity of the conditions during frequent observations led to the observations being interrupted for a short time, when they were resumed.

At 1 2′ P.M., at the right side there were sibili with inspiration

and expiration, and at the left side there were faint sibili, varied by occasional rhonchi, accompanying both inspiration and expiration. Crepitations were also heard at both sides during inspiration and expiration. The patient said his "chest was a little stiffer." The pulse was 96, and the respirations were 25 per minute.

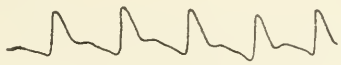


Fig. 25.—One hour and twenty-nine minutes after nitro-glycerine; pulse 96, respirations 25.

At 1 6′, however, while the crepitations continued, the rhonchi and sibili had again entirely disappeared; and the patient said his "breath was light again."

At 1 26′, at the right side there were slight sibilant rhonchi, and at the left side there were sibili and a few crepitations, with both inspiration and expiration. The breathing had become more difficult, according to the patient. The pulse was 84, and the respirations were 31 per minute.

Fig. 26.—One hour and fifty-one minutes after nitro-glycerine; pulse 84, respirations 31.

At 1 42′, the patient was again easier in his breathing, and the rhonchi and sibili had again disappeared.

Fig. 27.—Two hours and thirteen minutes after nitro-glycerine; pulse 84, respirations 26.

At 2, the only accompaniments at the right side were crepitations, but a slight sibilus occurred with crepitations at the left side. The patient stated that his breathing was "fairly easy."

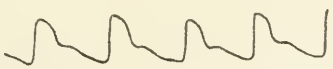


Fig. 28.—Two hours and twenty-four minutes after nitro-glycerine; pulse 82, respirations 24.

The breathing continued to be fairly easy until 5 P.M., when the sensations of tightness and difficulty reappeared; and it was found that rhonchi, sibili, and crepitations were continuous during the respirations.

Observation X.—The second observation in bronchitis, which I wish to describe, was made with nitrite of ethyl.

The patient, Annie M., 53 years of age, a washerwoman, was admitted into the Royal Infirmary on the 30th of December 1885, complaining of pain in the chest and side, difficulty of breathing, and constant cough with much expectoration. Two years previously she had suffered from some acute chest affection following exposure to wet. During the winter of 1884–85 she had suffered from a severe cold with cough, and the cough did not entirely disappear until summer. Four weeks before her admission she again had a cold, and her old troubles all returned. Treatment at home having produced little benefit, she applied for admission into the Royal Infirmary. When examined, she was found to be a well-built, strong woman, with an anxious, suffering expression. Her cough was frequent and violent, and was found to be associated with extensive bronchitis. There was a little emphysema, but no cardiac lesion could be detected. The expectoration was considerable in quantity, mucopurulent, and frothy.

On the 31st of December, the day following her admission, the symptoms had not materially changed.

At 1 15′ to 1 18′ P.M., over the front of the chest on both sides there were numerous sibili and rhonchi with inspiration and expiration. The pulse was 90, and the respirations were 20 per minute.

At 1 20′, she received two minims of a fifty per cent. alcoholic solution of nitrite of ethyl, diffused through two drachms of water.

At 1 22′, the only accompaniment heard with the breathing was an occasional brief rhonchus with expiration. She said her "breath feels easier."

At 1 23′, the breath sounds were entirely free from any accompaniment. The pulse was 90, and the respirations were 18 per minute.

At 1 24′, 1 26′, and 1 27′, this freedom from accompaniments continued without any interruption whatever.

At 1 30′, however, there were occasionally faint rhonchi at the end of inspiration, which, at 1 32′, were converted into sibili. The pulse was now 88, and the respirations sixteen per minute.

At 1 34′, the rhonchi and sibili had again disappeared, and they remained absent, and the breathing continued unembarrassed until 2 P.M., when the observations were interrupted. The patient was not again examined until 8 P.M., when she said the breathing had a short time previously become as difficult, and the chest tightness as great as it had been before she had received the dose of nitrite of ethyl. Rhonchi and sibili were found to be continuous over the front of the chest, and the respiratory movements were found to be laboured.

Observation XI.—The third observation I shall describe was

on a patient, Annie M., 19 years of age, who had suffered for some years from several diseases of the lungs, including pleurisy and bronchitis. She came under my care on the 9th of September 1886, complaining of pain in the chest, difficulty of breathing, and cough. Besides the symptoms of widely extended bronchitis, there was also evidence of old pleurisy, and of a lingering pneumonic inflammation at the back of the chest, both of which proved extremely refractory to treatment.

On the 11th of December 1886, at 12 50′ P.M., it was found that, at the front of the chest, there were at the right side sibili during inspiration, and rhonchi during the whole of expiration; and at the left side, sibili during inspiration, and harsh breathing with creaking sounds during expiration. The sputum was considerable in quantity, and consisted of rather viscid, frothy serum, having mixed with it a few masses of purulent matter. The pulse was 100, and the respirations 30 per minute.

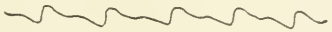


FIG. 29.—Two minutes before nitrite of amyl; pulse 100, respirations 30.

At 12 57′, the patient received four minims of nitrite of amyl, diffused through a little water.

At 12 57′ 30′, the face had become red.

FIG. 30.—One minute after nitrite of amyl; pulse 115, respirations 32.

At 12 58′ to 59′, at the right side there were only crepitations with inspiration, and a little creaking with a few crepitations with expiration; while at the left side inspiration was harsh, but without accompaniment, and expiration had only a little creaking at its termination.

At 1 P.M., the blush had almost disappeared from the face.

At 1 1′, the pulse was 99, and the respirations were 25 per minute. Rhonchi and sibili were still entirely absent.

At 1 2′, a sibilus occasionally was heard during inspiration at the left side.

At 1 4′, a similar accompaniment was now again heard at the right side.

FIG. 31.—Eight minutes after nitrite of amyl; pulse 99, respirations 32.

At 1 5′, however, neither sibili nor rhonchi were heard anywhere at the front of the chest, and they continued to be entirely

absent until 1 17′, although inspiration frequently became harsh, and crepitations and creaking sounds were generally to be heard.

At 1 19′, now and then a short sibilus accompanied inspiration at the left side.

At 1 20′, sibili were frequently heard during expiration at the right side.

Fig. 32.—Twenty-eight minutes after nitrite of amyl; pulse 106, respirations 30.

Rhonchi by-and-by added themselves to the sibili, until at 1 30′, or thirty-three minutes after the administration, the auscultatory phenomena had returned to very much the same condition as they had been before the patient had received nitrite of amyl. The pulse tracings show that the effects on the circulation were of much longer duration than on the dyspnœa and its associated auscultatory phenomena.

Fig. 33.—Forty-one minutes after nitrite of amyl; pulse 100, respirations 32.

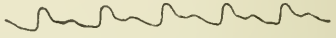


Fig. 34.—One hour and forty-three minutes after nitrite of amyl; pulse 92, respirations 32.

Observation XII.—In the next observation, the fourth in bronchitis, the effects of nitrites in bronchitis accompanied with an adhesive and scanty sputum are illustrated.

The patient, Alexander G., 49 years of age, had suffered, at intervals, for fifteen years before his admission into the Royal Infirmary, from bronchitis. Each attack was referred by him to a special "wetting" which he got while following his occupation as a shepherd. He had also had a long experience of rheumatic pains. He was a tall, well-built man; and his chief complaints were breathlessness, cough, frequently occurring in paroxysms of great severity, and great difficulty in expectoration. The lungs were found to be emphysematous, and to be affected with extensive bronchitis associated with a scanty, glairy sputum of small quantity. Nitrites were on many occasions administered to this patient, but the details of only two of these administrations will be here given: the first with nitrite of sodium, and the second with nitrite of ethyl.

On the 28th of November 1885, on examining the chest at 1 6′ P.M., sibili, now and then varied with rhonchi, were heard almost continuously with inspiration and expiration. The pulse was 56, and the respirations were 16 per minute.

At 1 9′ 15″, one grain of nitrite of sodium, dissolved in about a drachm of water, was given to the patient.

At 1 10′ 10″, the sibili and rhonchi had entirely disappeared, and

the breathing was no longer difficult. From this time until 1 52′, a period of forty-two minutes, frequent examinations of the chest showed that the breath-sounds remained absolutely free from rhonchi or sibili.

At 1 52′ 30″, however, faint sibili were heard at the end of expiration, and the patient said his breathing was "beginning to close up again."

At 1 55′, sibilus was frequent with expiration at the right side.

At 1 63′, rhonchus was present at both sides, and the breathing was as difficult as it had been originally.

Observation XIII.—An observation on this patient with nitrite of ethyl was made on the 24th of November 1885.

At 10 40′ A.M., it was found that over both lungs inspiration and expiration were accompanied with nearly continuous sibili, the pulse being 63 per minute.

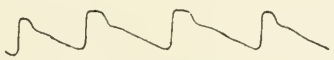


Fig. 35.—Immediately before nitrite of ethyl; pulse 66.

At 10 42′, two drachms of spiritus ætheris nitrosi (Phar. Brit.), estimated by an analysis to contain nearly two minims of nitrite of ethyl, were given to the patient.

At 10 43′, he said his breathing was easier.

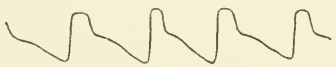


Fig. 36.—Two minutes thirty sec. after nitrite of ethyl; pulse 70.

At 10 45′, the sibili had entirely disappeared. The respiratory sounds remained absolutely free from accompaniments until 12 noon, or for a period of an hour and a quarter.

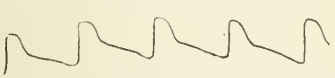


Fig. 37.—Five minutes after nitrite of ethyl; pulse 74.

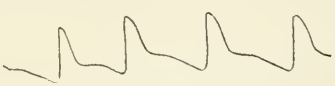


Fig. 38.—Thirteen minutes after nitrite of ethyl; pulse 72.

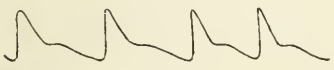


Fig. 39.—Twenty-three minutes after nitrite of ethyl; pulse 72.

Fig. 40.—Fifty-eight minutes after nitrite of ethyl; pulse 66.

The chest was not again examined until 12 30′, and then rhonchi were heard with inspiration and sibili with expiration.

Fig. 41.—One hour and forty-eight minutes after nitrite of ethyl; pulse 63.

These accompaniments were found to persist in subsequent examinations, while the breathing gradually became more difficult.

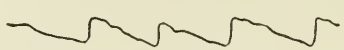


Fig. 42.—Two hours and eighteen minutes after nitrite of ethyl; pulse 60.

Observation XIV.—One of the best illustrations of the influence of nitrites on the dyspnœa and associated auscultatory phenomena of bronchitis was obtained in an observation on Thomas H., to whose case I have already referred when describing an observation made during a paroxysm of asthma which was treated with nitro-glycerine. I then alluded to the circumstance that several observations had been made on him when he was not suffering from asthmatic paroxysms, for the purpose of ascertaining the effects of nitrites on the symptoms of bronchitis, from which he also suffered during a portion of the time that he was under treatment in the hospital.

When the observation I wish now to describe was made, he had comparatively slight dyspnœa—the chief evidence of which was merely a sensation of weight and tightness in the chest.

At 1 20′ P.M., on the 12th of February 1887, it was found that at the right side inspiration was harsh, and accompanied with several coarse crepitations, and with occasional rhonchus; while the latter half of expiration consisted of a loud rhonchus. At the left side the conditions were the same, except that there were no crepitations, and that sibili and rhonchi occupied the whole period of expiration. There was no expectoration, nor had there been any during the previous two hours. The pulse was 75, and the respirations were 20 per minute.

At 1 30′ 30″, he received ten minims of a 10 per cent. solution of nitrite of sodium (1 gr.) diluted with a drachm of water.

At 1 33′, at the left side there were a few crepitations with inspiration, but no accompaniments with expiration; and at the right side the breath sounds were absolutely clear. The patient stated that the sensations of weight and tightness had disappeared from his chest, and that his breathing was "quite easy."

At 1 34′, the conditions of the breathing and of the breath sounds remained the same as at 1 33′.

At 1 35′, however, slight rhonchus was heard during a part of inspiration and of expiration over both lungs, and the patient said the breathing was "not quite so clear."

At 1 36′ 30″, he said the breathing was again "clear," and it was found that there were no longer any rhonchi at the left side, and only on occasions a slight rhonchus with inspiration at the right side.

At 1 37′, and at 1 38′ 30″, both sides were entirely free from any other accompaniment than a few crepitations, and the breathing was entirely unembarrassed.

At 1 42′ to 1 46′, brief rhonchi were occasionally heard with expiration, sometimes at the right, and at other times at the left side.

At 1 47′ to 1 55′, the breath sounds were again entirely free from rhonchi and sibili, and there was no dyspnœa.

At 1 57′, rhonchi were occasionally heard with inspiration and expiration at the right side.

At 1 59′, they had again disappeared, and they continued to be absent until 2 5′, when again a rhonchus or sibilus was heard at one or other side, and with either inspiration or expiration. No further change occurred until 2 13′, when these accompaniments were found to have disappeared, and they had not again returned at 2 40′, when the observations were stopped. While the accompaniments were absent there was absolutely no feeling of weight or tightness in the chest.

The effects on the pulse tension were rather slowly developed, but, as the three subjoined tracings show, they had not disappeared at the conclusion of the observation. There had been neither cough nor expectoration during the whole time following the administration of the nitrite.

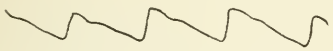


Fig. 43.—Before nitrite of sodium; pulse 76, respirations 20.

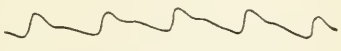


Fig. 44.—Twenty minutes after nitrite of sodium; pulse 78, respirations 19.

Fig. 45.—One hour and ten minutes after nitrite of sodium; pulse 73, respirations 18.

The last observation I propose to describe affords an illustration of the effects of a nitrite in bronchitis accompanied with profuse watery expectoration.

Observation XV.—The patient, Mary B., 30 years of age, became ill with bronchitis four months before her admission into the Royal Infirmary, in December 1886. She suffered, on admission, from palpitation, dyspnœa, and a frequent cough, with much watery and frothy expectoration. There was no disease of the heart, nor marked emphysema.

On the 16th of December, at 1 52′, rhonchi were heard profusely over all parts of the front of the chest, along with numerous small and medium crepitations.

At 1 54′, she received two minims of nitrite of amyl in one drachm of water.

At 1 57′, there were no rhonchi or sibili heard anywhere, and the patient stated that her breathing was much easier.

At 2 2′, the auscultatory phenomena and the breathing were the same as at 1 57′.

At 2 5′, however, rhonchi were heard at the right side, though only rarely. They, however, gradually became more frequent, and the breathing slowly reacquired its former dyspnœic character.

Including those that have now been described, sixty-one observations were made in bronchitis, of which detailed records have been preserved. In forty-eight of them the nitrite administered succeeded in removing every vestige of rhonchus or sibilus for various periods of time. In ten these sounds were lessened in their amount, but they were not altogether silenced. In only three the effects were either extremely slight, or altogether negative. The sixty-one observations were made on twenty-five patients suffering from bronchitis.

Whenever rhonchi or sibili associated with any sensation of dyspnœa were removed, the previously existing dyspnœa disappeared, or became much less marked during at least the time when the rhonchi and sibili were absent or lessened; but when nitrites failed to silence or to reduce the rhonchi or sibili they also invariably failed appreciably to lessen the dyspnœa.

It seems to me that the demonstration is complete that the dyspnœa of bronchitis, when associated with rhonchi and sibili, is mainly produced by the conditions of the bronchial tubes which produce the rhonchi and sibili. The cause of these sounds cannot be intumescence by congestion of bloodvessels or other results of inflammation, otherwise nitrites would increase, rather than lessen or suspend, the sounds. Adhesive mucus cannot frequently be a cause, otherwise nitrites would not, in so large a proportion of the observations, have succeeded in producing complete silence; for they have no special effect on deposited mucus, nor was it found that any particular influence was exerted by them upon the frequency of expectoration. The only explanation of the results of the observations that can reasonably be adopted, seems to be that in bronchitis the rhonchi and sibili are frequently produced by contractions of the bronchial muscles; that dyspnœa is produced by the impeded movement of air caused by the constrictions resulting from these contractions; and that both are removed by nitrites, because nitrites reduce the spasmodic contractions of the bronchial muscles.

I have been much gratified to find that the conclusions thus arrived at are in complete harmony with opinions expressed many years ago by my former teacher, Professor Gairdner. Writing, in 1853, on the subject of bronchitis, he states:[1] "As our information at present stands, we must confess ourselves to be unhesitating believers in the doctrine of spasm. We even go further, and think there is good ground for supposing partial

[1] *British and Foreign Medico-Chirurgical Review*, 1853, vol. xi. p. 477.

spasm to be in all cases connected with bronchitis, especially in its early stages, and to be the chief cause of that narrowing of the tubes at particular points, which is the most probable mechanical condition producing the sonorous and sibilant râles."

In a considerable number of instances nitrites succeeded in completely controlling the spasmodic contractions of the bronchial muscles for only brief periods of time, presenting a marked contrast in the duration of their beneficial effects in bronchitis to what was usually observed when they were administered in an asthmatic paroxysm. The explanation of this may probably be found in the circumstance that in bronchitis, as contrasted with asthma, the exciting cause of the spasm is a persisting and purely local one, which is not removed by the action of nitrites, but continues so long as the bronchial inflammation continues.

Still, in all but the relatively few instances where the effects were practically negative, it was found that relief was experienced for a long time after the rhonchi and sibili had returned; indeed, in many of the observations for several hours. The administration of a nitrite does not, therefore, require to be a very frequently repeated one; as the dry sounds, which sometimes quickly reappear, are still for a long time present only in a degree and amount which is much less than they originally possessed. It is, in most cases, unnecessary to administer the nitrite that is selected more frequently than every three or four hours.

Although nitrites dilate bloodvessels at the same time as they relieve the dyspnœa produced by spasm of the bronchial muscle, it has never occurred in these observations that they have increased the bronchial inflammation. Their action, when they subdued or lessened the dry râles, was invariably to give relief to the patient; in some cases this relief was apparent even on the day following that in which a single dose had been administered; and, in a few instances, bronchitis was altogether cured by their almost unaided influence. At the same time, I should anticipate that where marked tendency to bronchial or pulmonary hæmorrhage exists, they may increase this tendency, and, therefore, prove injurious.

I have not obtained any facts that would justify the assertion that any one of the nitrites is to be preferred in bronchitis, because it possesses therapeutic advantages over the others. There are, however, advantages of other kinds which lead me to give a preference to nitrite of sodium, and to nitro-glycerine. Each of these is stable, and can be used in solution, which admits of ready administration by the stomach, or by subcutaneous injection, in doses that can be accurately defined. Nitro-glycerine[1] may, therefore, be conveniently given, not only in the form of tablets of the British *Pharmacopœia*, but also dissolved in absolute alcohol, or rectified spirit, or distilled water; bearing in mind that a saturated

[1] The prejudices that are sometimes raised by the use of this word may be avoided by prescribing it under the name of "Trinitrine."

solution of 1 in 760 or 800 of distilled water can be obtained only by prolonged contact, that a solution in ordinary water slowly undergoes decomposition, and that solutions cannot be kept unchanged for many days in the presence of alkalies or alkaline salts. On the other hand, nitrite of sodium is freely soluble in water, and it remains unchanged for an indefinite time when dissolved in either distilled or ordinary water; but it is decomposed by acids, and for this reason it is quite possible that when the contents of the stomach are exceptionally acid in reaction the nitrous anhydride it contains may be so completely set free in the stomach that only a little nitrite will enter the blood—an accident, however, which could easily be prevented by giving it with an alkali.

Nitrite of amyl and nitrite of ethyl have not only the inconvenience of requiring alcohol to dissolve them, but also the great disadvantage of being very unstable substances, spontaneously undergoing change and deteriorating in the course of time. These inconveniences are fully recognised in the case of nitrate of ethyl, as it occurs in the *spiritus ætheris nitrosi* of the *Pharmacopœia*, which, however, has continued, notwithstanding, to be a favourite and widely used remedy. It is probable that the favour with which it is regarded is due not only to the action on the circulation which it shares with the other nitrites, and to its being an alcoholic preparation, but also to the previously unrecognised influence which it exerts on dyspnœa, when it is administered, as it so frequently is, in the treatment of bronchial catarrh.

The power of these substances to control bronchial spasm, whether that show itself in the orthopnœa of an asthmatic paroxysm or in the relatively slight dyspnœa of ordinary bronchitis, will probably lead to their being more largely used than they have hitherto been in the treatment of disease. Where their administration is successful in removing the auscultatory evidences of such spasm, it is difficult to imagine anything more convincing of the influence that may be exerted upon the conditions of disease by pharmacological agencies. The observer has presented to him a patient in whose thorax a continuous succession of varying sounds is being produced, and whose condition is one of distress, and sometimes of intense suffering and anxiety; within a few moments after a nitrite has been administered the conditions are entirely changed; the endless succession of noisy breath-accompaniments gives place to an almost complete silence, in which only the subdued quiet of the normal respiratory sounds is audible; and, at the same time, the distress of dyspnœa, or, it may be, the intense suffering and anxiety of urgent orthopnœa are entirely removed.

I have, in conclusion, to express my obligations to a number of gentlemen who gave me valuable assistance in these observations, and especially to Dr Sawers Scott, for some time clinical assistant, and to Drs Vaughan, Thompson, Robertson, Wilson, and Jeffcoat,

resident physicians, and Messrs Tofft, Traquair, Gibson, Dunlop, Loubser, Hawkes, Hutton, and others of the clinical clerks in my wards when the observations were being made.

As in each observation the chest was almost continuously auscultated, pulse tracings were taken every few minutes, the respiratory and pulse movements were frequently counted, and, in several instances, the movements of the chest and abdomen were recorded by means of Marcy's polygraph, while the observations generally lasted for one or two hours, it is obvious that any value they may possess has been largely derived from the assistance and co-operation of these gentlemen.

Prof. Gairdner, Glasgow, after having expressed his sense of the high value of Prof. Fraser's communication, remarked that he had no criticisms to offer. How could he, when Prof. Fraser had stated that the whole gist of his investigations was corroborative of what he (Dr G.) had written so many years ago? It might be interesting (in the fluctuations of opinion that have been in existence and pleaded in books) to the members of the Society to know that, having read most of the literature that had appeared on the subject, he had seen no reason to swerve from the idea that asthma was essentially a neurosis (a spasm, in fact); and he concurred with Prof. Fraser, that if they once admitted that view, then it followed as a matter of course that in bronchitis, which was no doubt essentially a catarrh or inflammation, the element of spasm must be regarded as almost constantly a part of the symptoms in each particular case. It was important to have his views confirmed as Prof. Fraser had confirmed them, and to find them made the indication for the application of therapeutic agents. At the same time, though he was unwilling to suggest that he had in any degree anticipated the admirable results of Prof. Fraser, yet his experience had led him in the same direction, and he might mention in illustration the case of an intimate friend, still a distinguished member of Edinburgh Society, who had been an asthmatic for a great many years, having had paroxysms innumerable at all seasons, and with intervals of good health, and had passed through all its phases. The lessons he drew from the close observation of this single case very many years ago appeared to point so irresistibly to spasm that it had remained rooted in his mind in a way that had never been got rid of, that the condition was a neurosis. Somewhere among the fifties he was called out one night to see this gentleman. Chloroform at that time was not new, but it was not much used except for its anæsthetic properties. It had, however, been found to give his friend relief, and the letter he received said, "Don't forget to bring the chloroform." He was a difficult patient to bring under chloroform, for it caused resistance and produced severe vomiting; but he noticed then what Professor Fraser had noted with regard to nitrite of amyl,

viz., that chloroform controlled the spasm absolutely, but only momentarily. When he got the patient into such a deep state of chloroform narcosis that a surgical operation could have been performed, all the râles immediately disappeared, but reappeared when he came out of it, thus contributing to rivet in the mind the idea of spasm as the chief, if not the sole, cause of the asthmatic paroxysm. Another fact tending to show him that asthma was not pathologically identical with emphysema of the lungs (which was then the doctrine of the French school), was that this gentleman, though very probably an emphysematous subject now, could scarcely be supposed at the time in question to have undergone more than the very first, though it was possible that there might have been the beginnings, if any, of emphysematous changes. His very first attack, and it was a very severe one, drove him home from his wedding tour. From this and all his earlier attacks the recovery within a few days, perhaps even hours, was usually complete. The night attack just mentioned, during which the chloroform was administered, was, from this point of view, a rather remarkable one, both from its extreme severity at the time, and from what occurred afterwards. He (Dr G.) had forgotten whether it was a Saturday or a Sunday, but the patient (who had by this time thoroughly learned his own disease so far that he knew that he was sure to be better in a couple of days) would not be restrained from coming into town for business on Monday, which was his board day. Knowing the severity of the attack, Professor Gairdner had interposed as well as he could, but it was of no use. He came into town and did his work, and did more. He set off that night for London. He crossed from London to the Continent, and without drawing rein he went straight on to Geneva, where he joined a friend who was a good pedestrian, and off they went to Zermatt, at the base of Monte Rosa, and within a few days he and his friend had walked over the Monte Moro pass, 10,000 feet above the level of the sea. If his asthma had depended on a congestive state of the air passages, one would have thought that this was the very thing to set it up, but it didn't. He might mention that this gentleman having once or twice spat up a very little blood, it was considered desirable to send him over with a letter to Stokes of Dublin, who said his lungs were perfectly healthy; and there could be little doubt that they remained so, or at all events not greatly emphysematous, for many years thereafter. If he were to put himself in the position for a moment of the advocatus diaboli, or counsel for the opposition, there was one question he would like to ask Professor Fraser. In one part of his paper he said that if the paroxysm of asthma were produced by a sudden congestive lesion, nitrite of amyl would certainly tend to aggravate it. On the general principle that nitrite of amyl dilated the arterioles, that was a tenable argument; but how would he dispose of it if put in this

form, that the systemic circulation being much more affected than the pulmonic by the arterioles, owing to the greater strength and resistance of their muscular walls, the effect was to relax the systemic, and thus allow the blood to flow in greater amount from the pulmonic circulation into it? The complete and sudden disappearance of the spasm, however, under amyl nitrite seemed to him to make it out of the question that there could be, as it were, a great tide of the whole blood flowing from one circulation into the other. The next point he would like to remark on was this. Every one must have noticed that the wheezing of bronchitis—the asthmatic element in bronchitis, as they might now call it—was very often seen to be in marked contrast, as regards its severity, with the more dangerous elements in a particular case. If they had in a pulmonary case one lung disorganized and the other not, it was almost always in the one not undergoing disorganization that these wheezing râles occurred. The same was the case when one lobe was becoming disorganized and the other not, or when the back of the lung was deeply implicated and the front was not. Could they take anything practical out of that observation? In the disorganized portion of the lung there was less tidal air, of course, and possibly the air vesicles were blocked, and therefore there was less of the wheezing, because there was less air to make the wheezing. But was that the whole explanation? Connect with it this other idea, even if it should at first appear gratuitous, or not worth considering. Why do we not all of us wheeze more or less in the healthy as in the bronchitic state? In the innumerable recesses of the bronchial tubes there must always be many chances for the air to get into vibration; and if those bronchial muscles were always liable to be set into spasm, was it not an extraordinary fact that healthy men went on from day to day without ever a wheeze existing in the chest? If the bronchi were rigid tubes on the one hand, or flaccid and non-muscular tubes on the other, it could hardly be but that the little obstructions from collections of mucus and extraneous particles would constantly, or at least very frequently, even in healthy men create currents of air that would induce wheezing at certain times and in certain parts of the tubes. The only conclusion he could arrive at was that the bronchial muscles were so beautifully regulated with respect to the function they had to perform, that there was an instant response through the reflexes in the medulla oblongata, by which they were placed in precise accommodation to the amount of air that was required to pass along the tube. This assumed function of the bronchial muscles, as regulators of the air-supply of particular lobules, had many attractions for one who adopts the views set forth by him in 1850–51, some of which at least might now be considered as part of the established doctrine received in medicine, that regarding collapse of the lung. It must be plain that in a great many different diseased states of the lung there

must be tendencies to collapse going on in various parts, and, in consequence, undue strain will be thrown on the lobules adjoining. Every such strain, if not met by some kind of opposing or regulating force, would, from the necessities of the case, tend to break up permanently a set of air-vesicles if carried beyond a certain point. It would lead inevitably to emphysematous dilatation, a condition which always destroyed the walls of the air-cells, broke up and ruined them, and so would be as destructive to their function even as tuberculization. He thought it a not unreasonable view that the bronchial muscles might be regarded as regulators to this extent, that by their contraction they saved, in some degree, those air-vesicles from the over-strain due to collapse of adjoining air-vesicles. But was that the whole function of the bronchial muscles? When he went into this he felt as if he were swimming in deep waters. If they accepted the view that everything in the body had a function, it was difficult to assume that the bronchial muscles were there only to produce the asthmatic spasm. It was on this account he was led many years ago to adopt the view that they were "scavenger muscles." By causing peristaltic action in the smaller tubes, they were constantly discharging outwards the mucus which was constantly forming and mixing with foreign matters from without, and which would otherwise tend to stagnate in the pulmonary apparatus just where it was most dangerous, He thought it a not unreasonable view, though it could not be proved, perhaps, by evidence that there existed some function of this kind which would be in accord with what they knew took place in the detrusor muscles of other hollow viscera. Take, for example, the pains of colic. What happened in these cases? What was intended to be expelled was retained so long as the spasm lasted. The expulsive action did not go on. A man suffering from colic might take any amount of colocynth pills, but they would not act till the spasm was over; and then he did not need the pills; the bowels would act without them. In a fit of asthma this deobstruent action was put a stop to. The considerable amount of mucus which normally would be forwarded into the large bronchi and trachea was retained in the smaller tubes by the interruption occurring to the normal "scavenger action." The moment the spasm was over, this retained mucus was discharged, and the first sign of relief was thus the patient's ability to expectorate. All this led him to ask another question, which lay more, perhaps, in Professor Fraser's province. What was an expectorant? Had there ever been any reasonable theory given of that class of medicines? Very shortly after he went to Glasgow in 1863, he read everything on this subject that he could; and the conclusion he came to was that there was not a word of common sense, and no reasonable explanation of the expectorant action of such medicines as squill, lobelia, senega, etc., to be found in any of the books; and if they did say anything, it was that one stimulated, another

depressed; one increased secretion, another diminished it, etc.; so that it would almost appear as if those complex old mixtures, which all of them knew were mostly composed of opposites, intended to neutralize one another, rather than of medicines co-operating towards one result. In the midst of this anarchy he was not surprised to find, in works of authority like that of MM. Trousseau and Pidoux, that the order of expectorants was dropped out altogether, and said to be non-existent. But if they adopted the view that the bronchial muscles acted in peristalsis and as deobstruents, then a clear and reasonable explanation of an expectorant was that it quickened peristaltic action. By acting on this particular function it changed irregular into regular action, and thus promoted expectoration.

Dr Allan Jamieson drew attention to the fact that Bulkley of New York had read a paper at a recent meeting of the British Medical Association on the connexion between asthma and diseases of the skin, and in it made observations on the theory of an urticarial condition of the bronchial tubes. Bulkley gave an analysis of a number of cases to bear out the view that there was a relationship between asthma and diseases of the skin, and especially urticaria. He tabulated sixty-eight cases of urticaria, and out of these only five had a coincident asthma. Dr Jamieson had gone over the records of about 2500 cases of skin disease, and in these found only a small proportion of cases of urticaria, so that it was not common, or very seldom came, to the specialist. In only one of these cases of urticaria was there asthma. This tended to support the view of Professor Fraser that asthma was not due to a congestion. In urticaria spasm was not prominent. The production of the white spot in the centre of the wheal had been a point of discussion. It was originally supposed to be due to pressure from the periphery to the centre, driving out the blood and causing the blanched appearance. Gull raised the question that it might be due to spasm of the small bloodvessels driving the blood out of the wheal. If so, the spasm was a very slight one. On that ground they must lay aside the view that urticaria and asthma were co-relative phenomena. The attention which would now be directed to the nitrites would probably lead to a largely extended use of them. Dr Broadbent had recently published a paper on the remote effects of remedies. He referred in it to certain cures of asthma. One of these was Himrod's. He said he was led to believe that when it was persevered in for a time it led to dilatation of the heart. Shortly after Dr Jamieson came on a case in the country where the patient, a young man, was in the habit of using Himrod's powder freely for asthma. After its use he became drowsy and livid. He was deprived of the Himrod, and ordered grindelia robusta, from which he got relief. While the nitrites were worthy of an extensive trial, a note should be added regarding the abuse of the remedies, because asthma was

a disease patients were apt to treat themselves, and they might be led to use the nitrites to such an extent as to cause permanent injury.

Dr Graham Brown said Professor Fraser had furnished a new clinical proof of the spasm that existed in asthma. There was no other clinical proof. The experimental proofs had been varied. The first was that of Volkmann. Then Paul Bert tied a tube into the trachea, and registered the movements of the air in the trachea while the vagus was stimulated. This was not a clear proof of the action of the bronchi, inasmuch as the movements might have been brought about by contraction of the whole lung tissue. After him Riegel passed a stream of air through the lungs of a living animal, making holes at the bases by which the air passed out into the open air. The pressure was registered, and on stimulating the vagus the air tension was increased; but he having started with the view that spasm of the bronchi was the cause of asthma, was led to throw over that theory, in part at least, in favour of spasm of the diaphragm, which he had observed to follow an exceedingly slight stimulation, and was led to believe it clinically of very much greater importance than bronchital spasm. He thought Riegel was wrong in that his methods were not likely to lead to definite results. In the experiments conducted by Professor Roy and himself they proceeded in a different way from previous observers. A glass tube with a bag of fine animal membrane fixed on its lower end was passed into the air passages, and its upper end connected with a registering apparatus. They got some rather curious information, which bore somewhat on the subject Professor Fraser had brought forward. On stimulating the vagus they got a powerful contraction of the bronchial tube. When they employed ether, on stimulating the vagus they got expansion instead of contraction, showing how valuable it might be in the treatment of asthma. If they arrested respiration, the bronchial wall in which their instrument was lying contracted sharply, showing dyspnœa itself aggravated bronchial contraction. They got some indications of a rhythmical bronchial contraction. For his own part he thought the bronchial spasm was a very important element in asthma, but he also thought spasm of the diaphragm was of great importance. In regard to the use of the nitrites, he wished to support Dr Allan Jamieson in calling attention to the dangers of using them without proper advice. The dyspnœa which had been the subject of discussion was a sort of preliminary dyspnœa, if he might so call it. If in an animal a contraction was brought about, there was a change in the rhythm of the breathing long before the blood was sufficiently venous to stimulate the respiratory centre. The first effect of constriction of the trachea was to bring into play the vagi, which acted so as to alter the rhythm and compensate the obstruction. When the respiratory centre was involved the tension of the blood rose. There must be some good reason for that elevation of the blood

pressure. If they gave nitrites during that period they did exactly the opposite of what the system was doing to repair the dyspnœa. The cautions were thus of importance. The spasmodic asthma must be pure. If there were any œdema the nitrites should not be given, because they depressed the blood pressure and vitiated the blood, altering it so that it was not competent to carry oxygen. This caution did not, of course, include cases of bronchitis in which the dyspnœa was due to spasm of the bronchial muscles.

Dr M'Bride asked if the vascular theory of asthma had been so thoroughly disposed of as had been assumed. This theory had originated with Weber, and been resuscitated by Clark. Clark said that in hay fever they often had a catarrh of the nose associated with asthma, and therefore by a series of arguments, drawn from analogy, he came to the conclusion that the same condition existed in the bronchi. One question was not touched upon in his argument, and that was whether there were in the mucous membranes of the bronchi an erectile tissue, such as had been proved to exist in the nose. He understood, from a high anatomical authority, that it was not altogether improbable that this tissue would be demonstrated, and if so, it would be difficult to controvert Clark's view.

Dr James Carmichael would have liked to have entered on the discussion of some points raised in the paper, but as time forbade it, he would content himself with remarking, that in using such remedies as the nitrites for the relief of bronchial spasm we were only treating, as we have hitherto been in the habit of doing with such remedies as chloroform, symptoms. He would like to ask Professor Fraser whether he considered the nitrites were curative of the organic condition on which the bronchial spasm depended, and whether he had made use of the remedies in cases of asthma associated with serious organic disease of the lungs or bronchial tubes? He had been interested in the allusion made by Professor Gairdner to pulmonary collapse associated with alterations in the calibre of the bronchial tubes. In a paper which he (Dr Carmichael) had read to the Society last spring on the Bronchial Catarrh of Children, he alluded to this subject, and mentioned cases of pulmonary collapse in young children which were recovered from, and which, in his opinion, could only be accounted for by the supposition of the existence of bronchial spasm.

Dr Affleck agreed with Professor Fraser in considering asthma a neurosis—a diseased innervation of the muscular fibres of the bronchial tubes. The case had been made out quite clearly for that as being one of the most important factors in the disease. He questioned, however, if he had given adequate prominence to the state of the circulation in the lungs as one of the causes of the dyspnœa. They saw asthmatic attacks occurring where the asthma was coincident with bronchial catarrh, where they could not get rid of the idea of extreme congestion. He was going to

have asked the same question as Professor Gairdner as to the effect of the nitrites on the pulmonic circulation. He wished also to ask if Professor Fraser had seen any immediate alarming effects from the use of the nitrites. He had tried nitro-glycerine for spasmodic asthma. In one case, after giving chloroform for several hours without much relief, he tried 1 minim of a one per cent. solution. It caused great pallor of the face, extreme depression, and alarming symptoms, which made him feel he could not repeat the dose. In this particular instance it might have been due to the patient having a large amount of chloroform. There was no flushing of the face, but pallor and extreme feebleness of the pulse.

Professor Fraser, in replying, said they had been deeply interested in the remarks with which Professor Gairdner had favoured them. He believed that physiologists were at one in attributing a regulative function to the bronchial muscles. He feared he must plead guilty to the charge that pharmacologists had not paid attention to the views that had been expressed regarding expectorants. Investigation, however, had not shown that there was any substance with such an action as Professor Gairdner had suggested. It was quite possible that the hypothesis Professor Gairdner had advanced, that the action of nitrites was different or less on the bronchial than on the general systemic circulation, might be put forward, but it was an hypothesis supported by no fact he was aware of. If a substance had a particular action on an artery in a limb when in contact with it, he did not see why it should have a different action on an artery in a bronchus. Besides, both vessels were equally parts of the systemic circulation. Dr Allan Jamieson, he observed, did not in his large experience of cutaneous diseases find any facts in support of the views set forth by Weber and Clark, that the dyspnœa of asthma was due to an urticarial intumescence. He quite agreed with him that caution was needed in the use of nitrites. They were active, and if there were complications existing, such as grave cardiac affections, they might give rise to inconvenient symptoms. This was, however, an objection that might be brought forward against any active remedy, such as digitalis, for example, which did a great deal of harm from indiscriminate use. In regard to what Dr Graham Brown had said, he had made some reference to the knowledge that existed as to the contractility of the bronchial tubes. He should be inclined to lay much stress on the observations of Williams, who had made the bronchial tubes contract to such an extent as to obliterate their lumen. He understood Dr Brown also to say that the dyspnœa in these observations had not reached any extreme degree. That was not quite the case. In some of the paroxysms there was lividity of the countenance and feeble pulse, and the paroxysms had sometimes lasted three quarters of an hour, and still nitrites were successful. As to spasm of the diaphragm, he had refrained from saying it did not exist. He

wished to distinguish between primary and secondary causes and stages of dyspnœa. The whole respiratory apparatus was stimulated, and thus the diaphragm and all the auxiliary muscles were implicated, in the later stages. He believed the diaphragm had no more to do with the dyspnœa than could be explained by its being a very powerful muscle of respiration. The action of nitrites on hæmoglobin could scarcely be a cause of danger, unless enormous doses were given. Such doses as he had referred to would not produce methæmoglobin sufficiently to give rise to an exaggeration of the dyspnœa. In regard to Dr Affleck's observation, he thought the alarming result he mentioned was perhaps to be explained by the explanation he himself had given. It was a well-known fact that the prolonged administration of chloroform lowered the blood tension. When a great lowering had been produced, if they administered anything which dilated bloodvessels, it would not be wonderful if syncope were produced. Dr Carmichael had asked him a question as to whether this were a curative method of treatment, or if it merely gave relief. It could be claimed that it not only gave relief, but that in pure asthma it might even be curative. They might stop and practically cure the asthma which recurred periodically, if the periods were broken by the administration of nitrites. In any case, the relief meant a great deal to the patient.

Meeting VI.—April 6, 1887.

Professor GRAINGER STEWART, *President, in the Chair.*

I. ELECTION OF NEW MEMBER.

J. F. Sturrock, M.B., Golspie, was elected an Ordinary Member of the Society.

II. EXHIBITION OF PATIENTS.

Mr A. G. Miller showed—(*a.*) A new method of DRESSING for cases of circumcision. It consisted in first closely suturing the remains of the prepuce with from eight to ten catgut sutures, then painting the surface with Friars' balsam and covering it over with two or three plies of cotton wadding, on which the balsam is poured. The glans penis was left sufficiently free to allow of water passing. The band or ring of dressing should be at least one inch broad. The dressing was not suitable for young infants who were frequently wetting. In the case of older children they might be allowed to go about on the second or third day, when the dressing would be quite dry, and would not require to be changed or renewed. The case shown was a very good test of the method, as the operation had been done by the house-surgeon for

paraphymosis with some ulceration. The dressing was removed ten days after, and the wound was perfectly healed. (*b.*) A lad who had EXCISION OF THE RIGHT KNEE performed on the 25th June for gelatinous degeneration with destruction of cartilage, and was discharged on the 20th of August. The operation was done through two semilunar incisions enclosing an elliptical portion of skin removed to prevent redundancy. Bony ankylosis had taken place, and the shortening was very slight, the one boot being worn a quarter of an inch higher than the other.

III. Exhibition of Pathological Specimens.

1. *Mr A. G. Miller* showed the parts and attached skin from a case of EXCISION OF THE KNEE, to show how much skin was removed. The operation was done on the 8th March for ankylosis with old-standing disease. The wound had been dressed in all about five times, and was virtually healed. In this case and that of the lad shown above there was no redundancy or overhanging of the skin as often occurred when the ordinary simple semilunar incision was employed and no skin removed.

2. *Dr James Ritchie* showed a STOMACH and a PORTION OF LIVER to which it was adherent. The stomach exhibited in a very marked degree hour-glass contraction. On the upper and posterior portion of the pouch next the cardiac orifice was an ulcer, the base of which was formed by the liver. During an act of retching, four days before death, part of the attachment gave way, permitting a very small quantity of the gastric contents to pass into the peritoneal cavity, where they produced circumscribed inflammation. There was a degree of matting in the upper part of the abdomen, and both surfaces of the liver were covered with a layer of pus. The gall-bladder was small, being firmly contracted round a single stone.

3. *Mr Cathcart* showed an EPITHELIOMA OF THE SUBLINGUAL GLAND which he had removed through the floor of the mouth. The submaxillary gland, which was much indurated, had been removed at the same time. The patient had been quite unconscious of the growth in his mouth until eight days before his admission to the Infirmary, and even then it had been his medical attendant who had drawn his attention to the swelling. When he came for advice to the Infirmary a firm elastic swelling could be felt in the position of the left sublingual gland, with considerable induration about the submaxillary gland. On the floor of the mouth were some raised granulations surrounding what seemed to be an enlarged orifice of the submaxillary or of the sublingual ducts; from this granular *débris* could be pressed out. There had been some doubt as to the exact diagnosis, but as the tumour seemed to

be undoubtedly malignant, both submaxillary and sublingual glands were excised. A fresh section of the latter showed epithelioma. Parts of both were being hardened for future study. Probably the disease had started at the orifice of the salivary ducts and had spread inwards. If it turned out that the submaxillary gland was also the seat of epithelioma, the simultaneous passage of the disease along the respective ducts, from the place where they open close together, would explain this double onset, which would otherwise have been a difficulty.

4. *Dr Skene Keith* showed two FIBROID TUMOURS. The first was removed by Dr Keith on the previous day from a patient of Dr James Young. She had ceased to menstruate for two years and ten months, and the tumour had not decreased. No fewer than eighteen vessels, each of them as large as the radial, were seen passing into the tumour. In the uterine cavity, which was very large, were two polypi, and the entrance to the vagina could not be made out. The second, which was much smaller and with a point of attachment which could be covered by the thumb, had been removed by Dr Keith that forenoon. There was a very firm band of attachment binding the tumour down into the pelvis. If the corkscrew had been used, the band would have been torn through, and as it lay over the iliac vessels, some serious damage might have been done. The patient was only twenty-eight years of age.

IV. EXHIBITION OF INSTRUMENTS.

1. *Dr A. James* showed an INSTRUMENT for paracentesis of the chest, which was a combination of the aspirator and syphon. To an ordinary aspirator syringe was fixed a T-tube, having a stopcock at either free end. Rubber tubes passing from these were connected, the one with a safety trocar, the other with a weight. The instrument could be used either as an aspirator or as a syphon, and would, he believed, be an improvement on the ordinary forms of aspirators at present in use. It was also cheaper in price.

2. *Dr MacGillivray* showed a new form of PHYMOSIS FORCEPS he had designed for the removal of the plebeian and indurated prepuces usually met with in the Lock ward. The blades were parallel, fenestrated, and ribbed, their upper parts being spiked so as to take a firm hold of the portion of the organ to be removed. A knife passed through the fenestrum or slit in the blades, removed the part, leaving a cleaner cut than by any other method. It was not, however, necessary for the infantile and aristocratic prepuces which occurred in ordinary practice, dressing forceps being, as a general rule, all that was required.

3. *Dr Felkin* showed a Martin's PATENT SCREW FASTENER, which was very useful for hanging diagrams. It was an ordinary pin bent

a little way from the point into a spiral. It fixes diagrams easily and firmly to both wood or cloth.

V. Original Communication.

Dr William Hunter then read a paper on THE PATHOLOGY OF BLOOD-DESTRUCTION WITHIN THE LIVER, based on a long series of observations and experiments with which he was still engaged.

The evidence pointing to the liver as an important seat of blood-destruction under physiological circumstances was reviewed, and attention directed to the value to be attached to the presence of blood pigment within the various organs concerned in blood-destruction, viz., the liver, the spleen, and the red bone marrow.

The mode of destruction of red blood corpuscles within the liver differed entirely from that observed to occur elsewhere.

In the spleen and bone marrow the destruction of red corpuscles was a process of digestion on the part of the numerous cells there present, the corpuscles being either taken up bodily within these cells, or acted on by them from without. The result of this action was the formation of blood pigment in the form of larger or smaller granules of golden yellow colour, consisting for the most part of iron, most probably in the form of albuminates. Such granules, therefore, gave all the reactions of free iron, *e.g.*, becoming black when treated with sulphide of ammonium, blue with ferrocyanide of potassium and dilute hydrochloric acid.

In the liver, on the other hand, the destruction of the red corpuscles was in all probability effected through the action of the bile acids secreted by the liver cells—not, as had hitherto been supposed, by the action of leucocytes within the capillaries. The result of the breaking up of the hæmoglobin was in part the formation of bile pigments and the liberation of free iron, both constant constituents of the bile.

The essentially chemical nature of this disintegration explained the absence of any blood pigment particles within the liver cells in health. Hence, while in health the tissue of the spleen and red bone marrow gave a slight reaction of iron with the above reagents, that of the liver gave none.

Under abnormal conditions, however, the amount of pigment found in these organs might be considerably increased. Thus after transfusion of blood in rabbits, the amount of pigment in the spleen and bone marrow was found largely increased, and pigment in very slight amount was found in the liver cells. In the case of rabbits the important result appeared, that the subsequent health of the animal after the transfusion seemed in some degree to depend on the relative amount of blood-destruction which had taken place in the spleen and bone marrow on the one hand, and the liver on the other. Improvement in health seemed to be always associated with the presence of a large amount of blood pigment

in the former, while some degree of ill-health was usually associated with the presence of an excess of pigment in the latter.

These observations were of special interest in their bearing on the pathology of certain forms of idiopathic anæmia, *e.g.*, pernicious anæmia, in which disease a large excess of blood pigment within the liver was the one constant pathological condition found present. The view usually held, that this pigment was simply in process of removal by the liver cells, and had been conveyed thither from other organs of the body or from the blood, was shown not to correspond with the appearances presented either by the blood or by the other organs of the body in that disease; and hence the conclusion seemed unavoidable, that excessive destruction of blood corpuscles on the part of the liver was a far more important factor in the production of such forms of anæmia as were characterized by their perniciousness than had hitherto been considered probable or even possible.

Attention was further drawn to the fact that the rapidity of blood-destruction—after transfusion at least—as determined by enumeration of the blood-corpuscles, seemed to depend much more on the activity of the liver than on that of the other blood-destroying organs. This was evidenced by the richness of the liver in iron in cases in which the removal of red corpuscles had been specially rapid. In this relation the appearances presented by the liver after transfusion of blood mixed with a 5 per cent. solution of phosphate of soda, as recently advocated, were of special interest—his experiments showing that the injection of this salt along with the blood hastened the destruction of the red corpuscles, as evidenced, not only during life by careful enumeration, but also after death by the excess of iron found within the liver. Some clinical observations already made after such transfusions seemed to be in entire accordance with these experimental results.

Dr P. A. Young said he had at one time paid a good deal of attention to this subject, and had written his thesis upon it. It was very interesting to observe how it had gone on developing since then. They knew, or at least guessed, that the liver was a blood destroying organ. The presence of iron in the bile was one of the things which indicated this. He was, he believed, the first to calculate the amount of iron in the bile. From the amount of iron the hæmoglobin could be calculated, and from the hæmoglobin the number of corpuscles. The iron excreted by the liver was not entirely lost, but the greater part was reabsorbed into the system again, to be taken up by the hæmoglobin. The phosphate of soda, acting as a liver stimulant, destroyed blood corpuscles, and thus, by colouring the motions when there was suppression of bile, appeared as if it increased the bile flow, when in reality it only stained the fæces by the products of blood-destruction. This might be a source of error in judging of the action of so-called

liver stimulants. Could Dr Hunter tell them if other liver stimulants, such as iridin, acted in the same way?

Dr Foulis asked whether, seeing that phosphate of soda acting as a liver stimulant destroyed blood corpuscles, its administration to an anæmic patient, as was done in transfusion, was really beneficial?

Dr Hunter, in reply to Dr Young's question, said he had not as yet experimented with iridin and other hepatic stimulants as with phosphate of soda. The great difficulty attending the use of such drugs for purposes of transfusion was that the solvent used frequently itself acted on the red blood corpuscles. Any effect the drug might have in causing an increased destruction of blood corpuscles could not then so readily be ascribed to its action on any particular organ, *e.g.*, the liver, as in the case of a salt, *e.g.*, phosphate of soda, a solution of which (5 per cent.) had no local action at all on the red corpuscles. As to the use of phosphate of soda in transfusion, he considered it would ultimately be found of little value, more particularly in cases of pernicious anæmia, in which, as the observations just recorded seem to show, an excessive destruction of blood corpuscles on the part of the liver was already going on. The effect of the injection of phosphate of soda along with the blood was, as had been seen, to stimulate the liver to an increased activity. Clinical evidence in support of this peculiar action of phosphate of soda after transfusion could also be adduced, cases having occurred in which, instead of a rise, there was an actual fall in the number of corpuscles on the day following the transfusion. Of great interest in this connexion was the occurrence of such cases as the one related by Dr Milligan, in which, after a transfusion of blood along with phosphate of soda in a woman suffering from great loss of blood, well-marked jaundice developed in the course of the following 24 hours. This result would readily be explained by Stadelmann's observations, which went to show that the increased formation of bile pigments which followed on an excessive destruction of hæmoglobin on the part of the liver, was generally, in the first instance, accompanied by greater concentration of the bile, and a tendency for the bile to stagnate in its flow along the bile ducts.

Meeting VII.—May 4, 1887.

Dr Blair Cunynghame *in the Chair.*

I. Pathological Specimens.

1. *Dr G. S. Woodhead* showed—(*a.*) Portions of diseased lungs exhibiting tubercular invasion of the bronchi. In tubercular

children one often found large masses of tubercular glands lying along the bronchi, and more especially at the bifurcation of the trachea. These were frequently softened, and were in many cases simply caseous masses in the centre, with a firmer surrounding cortex. In some cases there was no invasion of the wall of the bronchus; but in others, small tubercular nodules might be seen in the mucous membrane near the bifurcation, and in one of the specimens exhibited they had a very interesting condition. This was a case in which the glands near the trachea and bronchi were enormously enlarged and much softened in the centre. On slitting up the left bronchus, an ulcer was found communicating with one of these softened glands just at the bifurcation. At this point there had been formed an opening in the wall of the bronchus, and a communication had been set up between the left bronchus and the shell of a gland immediately outside its wall. Through this opening the caseous material had evidently been emptied into the bronchus. In the lung two sets of changes might be distinguished. There was, first, a series of comparatively chronic tubercular nodules, very small and not well defined (chronic interstitial or lymphatic tubercle), and, second, a catarrhal or broncho-pneumonic tubercle, set up by the material derived from this caseating gland, which had been drawn in to the terminal air vesicles. In the injected lung they saw a beautiful mapping out of these lobules, as there had been in them an obstruction to the flow of the injecting material. These two forms of tubercle had evidently a very different life history; their structure was different, and the process of degeneration also differed very markedly. (*b.*) MICROSCOPIC SECTIONS OF THE KIDNEY, SPLEEN, AND LIVER, from a case of acute purpura hæmorrhagica. The case was that of a tramp, who died within thirty-six hours of his admission to Leith Hospital. A number of minute hæmorrhages were found under the skin, serous membranes, and in other parts of the body. Sections through these, and of the various organs, showed, after staining by Lœffler's method (alkaline methyl blue), masses of micrococci. These micrococci could also be stained very brilliantly with picro-carmine. He hoped that Dr Male, who had charge of the case, would before long communicate a full report to the Society.

2. *Dr Bennet* showed a PORTION of the FRONTAL BONE removed by the trephine on account of a punctured fracture. The patient, a boy of 4, was brought on foot to the Leith Hospital, where a small transverse wound above his right eyebrow was dressed, after which he walked home. Within an hour he was brought back unconscious, with a history of having had a convulsion. When Mr Bennet saw him he presented the usual symptoms of compression combined with those of cerebral irritation. The wound was enlarged and the trephine applied. As soon as the piece of bone including the fracture was removed, the boy became arti-

culate, and had done well since. The fracture had been caused by a stone thrown at him.

II. Exhibition of Instrument.

Mr Cathcart showed an ADAPTATION to the SILVER CATHETER used for retention from prostatic enlargement. It consisted in a little round head large enough to fill up the eye of the catheter, and attached to the end of the wire stilette. This prevented any blood from entering the instrument while it was traversing the urethra. It also had the advantage that the urine did not begin to flow till the wire was completely withdrawn, giving time for the adjustment of the vessel which was intended to receive it.

III. Original Communications.

1. MELANOTIC SARCOMA, PRIMARILY OF THE SKIN, SECONDARILY OF THE BRAIN AND OTHER ORGANS.

By W. Allan Jamieson, M.D., F.R.C.P. Ed., and Byrom Bramwell, M.D., F.R.C.P. Ed.

Many examples of multiple melanotic sarcoma of the skin have been recorded, notably by Kaposi, Hardaway, and G. H. Fox. Hardaway has illustrated his cases by two excellent chromo-lithographs, in the *Journal of Cutaneous Diseases* for 1883, 1884, and Fox represents an instance where the tumours could be counted by hundreds in his photographs of skin diseases. But solitary melanotic sarcomata are much rarer. In all my medical experience I have met with but two. Of the first the notes are unfortunately incomplete.

Mrs A., 40 (?), Berwick-on-Tweed, consulted me in August 1872. She stated that a year before, Dr Fluker had removed by ligature a small pedunculated tumour from her back. When she came to me there was a nodulated growth the size of the fist on the left side of the spine, in the dorsal region. It was dark purplish-black in colour, and bled readily. The entire mass was excised, all the tissues being cut away to the fascia beneath. Examined microscopically, it exhibited a locular structure, a fibrous matrix enclosing large cells filled with brown pigment. The tumour apparently originated in the substance of the true skin. The wound healed, and she continued fairly well for some time, but a similar growth developed subsequently in the right axilla. This enlarged, broke down in the centre, and discharged bloody serum and black *débris*. She eventually sank, but the date of her decease was not noted, and no post-mortem was obtained.

The subject of the second case was from time to time under my observation during nearly six years, and in preparing the account I have had the valuable aid of Dr Byrom Bramwell, who has

contributed the pathological investigation,—a work in this case of no ordinary trouble.

S. E. M., 26, unmarried, was sent to me in 1881 by Mr Chiene. She was a fair woman, not very robust in appearance, though well nourished. Skin fine and thin. She had once some hæmoptysis, and suffers every winter from bronchitis. Since ever she can remember has had a black mark on the right shoulder, just above the spine of the scapula. It was, when she was ten years old, rather less than a threepenny piece in size. She cannot tell whether it was congenital or not. On one occasion she vexed her father, and he struck her sharply on the shoulder with a stick, just where the pigmentary nævus was. Since then it began to enlarge, and when I first saw it, it had reached the size of a half-crown piece. When looked at it appeared little if at all elevated above the skin level, but on passing the finger over it, small shot-like bodies could be felt to move. At the upper part it was a deep bluish-black in colour; the cuticle looked thin, and as if loosely attached, and wrinkled vertically in fine lines. Here, too, it was sharply defined, while at the lower part there was an outlying ring, reddish-brown in hue, which enclosed an area of almost normal skin. The mark was quite superficial, and apparently involved little else than the epidermis. It itched at times, and was painful when dragged, not when pressed. A half-brother on her father's side has a brown pigmentary mole on the left leg near the ankle, accounted for in his case by his father having seized his mother suddenly by the leg, in the same situation, while pregnant.

The mark on S. E. M.'s shoulder was scraped out. It resisted the spoon, but was all removed except a small remnant of pigment. As I was somewhat doubtful of the exact nature of the mark, I submitted the scrapings to Dr D. J. Hamilton, who carefully examined them, and pronounced it to be a blood-clot under the skin. The discs were quite distinct, and had continued, he thought, unabsorbed. When seen, six months after, some nodules of keloid had developed in the scraped part; these were rather tender, and even spontaneously painful. They were treated for a time with salicylic acid collodion, without much, if any, good result.

She was not again seen for nearly four years. She then stated that she had continued well till two months before, when a change took place in the state of the shoulder, which had assumed once more its black pigmented appearance, and something more. There was now a fungating black nodule, bleeding a little at one part, and the size of a walnut. Round it the skin was smooth and pliant, though stained brown. She looked depressed, and her complexion had become sallow. With the assistance of Dr Alexander Bruce, I removed the tumour and pigmented skin, including also a margin of healthy tissue all round. The portion originally scraped was now white and scarlike. This was also taken away. The wound healed partly by first intention, but more by granulation, and in

the scar and stitch marks keloid developed, which, however, after a time became absorbed, and the cicatrix eventually was smooth and thin. The tumour on examination was found to be a round-celled melanotic sarcoma. It had been cut out down to the fascia, apparently quite below its deepest part; yet Dr Bruce found traces of the growth even at the lowest edge of the sections, and predicted a recurrence. The tumour was removed on the 23rd of October 1885.

With exception of an attack of bronchitis last spring, she remained fairly well till the 22nd of July. On that day, while menstruating, she went out into the cold and rain bareheaded, because her head ached, as it had done at times of late. She now began to see bright flashes of light before her eyes, and in association with these there occurred pain in the head. She stated that the flashes of light were first noticed one evening as she was reading the *Evening News*. They were worse, she thought, in the right eye. They resembled in colour and brilliancy the electric light, and the bright appearance which was seen seemed to vary in size from a small button to a large globe. The flashes were always accompanied or followed by headache. They at first occurred very often. She said she saw quite well between the attacks of headache, but was completely blind during the paroxysms.

I saw her on the 6th September 1886; she was in bed, and complained of great sickness and intense headache. She had not again menstruated, and there was a possibility that she was pregnant. She had severe and sudden attacks of sickness and retching.

When the pain in the head came on, the symptoms were as follows:—Flushing of the face, then intolerable cephalalgia, which lasted three to five minutes, then subsided. The pulse, however, became quickened and irregular during the attack; at other times it averaged from 66 to 72, and was regular. She could not bear to be moved, would lie thirty-six hours on one side at a time, so much afraid was she of reinducing the pain. She had some difficulty in directing the movements of the left arm, but had no real uneasiness, except in the head, and a little in swallowing. She was seen by Dr Bramwell on the 27th, and also on the 28th of September. He reports as follows:—

"Visual acuteness was not tested, but so far as could be ascertained by rough measurement (*i.e.*, without a perimeter), there was no contraction of the field of vision; there was certainly no well-marked hemianopsia. Well-marked double optic neuritis was seen with the ophthalmoscope.

"*Condition during Attacks of Headache and Flushing.*—I found the patient very much flushed in the face, and perspiring. She was lying perfectly still with the eyes closed, and suffering, the nurse said (and as she herself, after the paroxysm was over, explained), from intense headache. The pain was said to be

much worse on the right than on the left side. The pulse was slightly intermittent, its exact frequency during the paroxysm was not ascertained, for while the foregoing points were being observed, and before the pulse frequency was counted by the watch, the paroxysm of headache and flushing had passed off. After the paroxysm the pulse became smaller and more frequent, and numbered 100 in the minute.[1]

"*Condition of the Reflexes.*—The plantar reflex was lively on each side; the knee-jerk could not be elicited on either side. The muscles of the lower extremities were soft and flabby.

"*Mental Faculties.*—The mental faculties were quite clear; there were no emotional derangements or other symptoms suggestive of hysteria.

"*Relationship of Headache and Flushings.*—The headache and flushing were always observed in combination; that is to say, she never had a paroxysm of headache without flushing, and never flushing without having headache.

"*Paralysis.*—None at that time."

Dr Bramwell confirmed my diagnosis of a cerebral tumour.

It ought to be noted that the nurse on several occasions made use of the thermometer before, during, and after a paroxysm, but the temperature did not rise above the normal standard.

At this time she had as many as twenty attacks of pain during the night, and thirty in the day. She could see; taste and smell were normal, or at least not abolished. Hearing was acute, even too much so. She had no giddiness, though as she never was out of bed, this could not be absolutely determined. Certainly there was no complaint of giddiness when recumbent, but her alarm at the mere mention of being moved was so great, in case the act of motion should reinduce the intense pain, that she lay without venturing to stir a limb. When the pain was present she insisted she was blind; but as she kept her eyes resolutely closed so long as the agony lasted, this, too, was unconfirmed. On one day, the 29th of September, the pulse fell to 54, and on that day the attacks of pain were exceptionally numerous. On the following day the pulse rose to 90, and then there were twenty attacks of pain in the twenty-four hours. Hypodermic injections of morphia (Wyeth's tabloids) relieved the pain very considerably, and were repeated usually twice a day. Towards the end of October, she lost, very gradually, power of motion of the left arm and left leg. There was no loss of sensation before loss of motion ensued; but she had seldom, and but little, used that arm for long before. She emaciated greatly, then control over the sphincters was lost. There

[1] I had only one opportunity of examining the patient during a paroxysm of headache; my notes (made on returning home after seeing the patient) state that the pulse frequency was diminished during the attack; probably this was a mistake, for Dr Jamieson, who repeatedly observed the paroxysms, always found the pulse frequency increased during the attack. We have, however, thought it right, as this discrepancy in statement exists, to record it.

were no spasms, no convulsions. The urine throughout was free from albumen. It was not tested for melanin, but was never noticeably dark or smoky. She retained consciousness till the last. The temperature rose to 100°·8 on the 1st November, and she died with symptoms of pulmonary congestion on the 2nd.

There are one or two points connected with these cases which deserve attention. One of these is the origin of the tumour in the latter instance in a mole, a circumstance which has been observed before. Babes, in v. Ziemssen's *Handbook* says, "Melano-sarcoma of the skin usually develops from pigment moles, more rarely primarily." Again, this malignant alteration is not uncommonly secondary to a traumatic lesion. Thus Schuchardt (Volkmann's *Klinischer Vorträge*, No. 257), "It constantly appears that, while long-continued, frequently repeated slight irritations favour the development of carcinoma, a single severe injury (trauma) is most often the external exciting cause of a sarcoma. He cites two cases of Volkmann's:—1. A young girl of 20 received a blow with a stick over the right eye. An ecchymosis and inflammatory swelling formed; the latter did not become absorbed. From the roof of the orbit a medullary sarcoma arose; and though promptly removed, was followed by death due to general metastatic growths. 2. A girl of 16 or 17 fell on the street and struck the head of the tibia against the curbstone, and was carried home. A severe contusion with ecchymosis resulted. In this there rapidly developed a medullary sarcoma. Amputation was resorted to; but similar tumours formed in the lungs and cranium, and death within four to five months. The preliminary ecchymosis in both these is interesting, for in S. E. M.'s case Dr Hamilton found in the scrapings only unabsorbed blood-corpuscles—no sarcomatous elements. It was this report which determined me not to remove the whole by excision after dealing with the nævus by erasion. Whether a more radical mode of treatment would have saved my patient eventually is uncertain, as in neither of the two cases recorded by me did any recurrence take place in the primary seat of the disease. The scar remained healthy. The secondary deposits occurred in remote parts. There were peculiar mental influences at work in the second of my cases, much distress of mind, and other painful circumstances, which cannot be more than hinted at here, but which unquestionably exerted a depressing effect, and predisposed to the development of a malignant growth.

Post-mortem Appearances.—The post-mortem examination was made on 4th November at ten A.M.

The body generally was thin, but there was a fair quantity of fat in the anterior abdominal wall and over the internal viscera.

The skin surrounding the cicatrix, which had resulted from the removal of the melanotic growth described above, and the cicatrix itself, were perfectly normal and free from any melanotic deposit.

Head.—The scalp, skull cap, and outer surface of the dura mater were normal.

The inner surface of the dura was also natural except at one point, corresponding to a point just above the tip of the left occipital lobe, where a melanotic growth the size of a walnut was situated; it was adherent, on the one side, to the surface of the brain, and, on the other, to the inner surface of the dura. The portion of the dura which surrounded the new growth was considerably thickened and infiltrated with sarcomatous elements.

The convolutions of the brain were markedly flattened and the sulci effaced; the surface of the brain was very anæmic.

The pia mater and arachnoid were normal, and there was no appearance of meningitis.

Several small melanotic deposits were seen here and there on the surface of the hemispheres; the largest, which was about the size of a cherry, was situated at the anterior end of the inferior right frontal convolution.

The brain was removed whole, injected with Müller's fluid, and reserved for subsequent examination.

The optic nerves appeared natural; the right disc, which was removed for the purpose of microscopical examination, presented the usual appearances of well-marked papillitis; the left disc was not examined.

Other Internal Viscera.—The right tonsil was enlarged to twice its natural size, and infiltrated with melanotic growth. The glands on the right side of the neck were free from disease; one in the middle of the left side of the neck was as large as a pigeon's egg, and was absolutely black throughout from melanotic deposit.

There was some recent congestion and œdema of the lungs; but the thoracic viscera were otherwise perfectly normal.

Two melanotic deposits, the size of large marbles, were found in the omentum; several melanotic tumours, the largest the size of a large walnut, were adherent to the outer surface of the intestine, opposite the mesenteric attachment.

There were several small melanotic deposits, some of them wedge-shaped, and in size and position resembling infarctions, on the surface of both kidneys.

The liver and spleen were healthy. The uterus contained a three and a half months' fœtus.

Subsequent Examination of the Brain.—When the brain was sufficiently hardened, it was cut into a series of transverse vertical sections, which were photographed (see Figs. 1 to 7).

In this way numerous deposits of melanotic sarcoma were displaced in the cerebral tissue. It is unnecessary to attempt to describe the exact position of each separate melanotic mass. Suffice it to say, that the cerebellum and the pre-frontal and occipital lobes were the parts which were chiefly invaded. The motor regions of the brain and the great basal ganglia were much

less extensively involved; a few minute, almost microscopical deposits were present in the lenticular nuclei; a nodule of larger

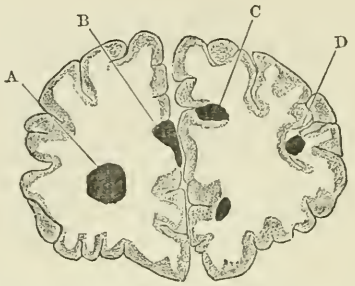


Fig. 1.—*Transverse Vertical Section through the Frontal Lobes in the case of S. E. M., showing Melanotic Deposits* (A, B, C, D) *of some size.* (Reduced from a Photograph.)

size involved corresponding and adjacent portions of each optic thalamus; and a small nodule was situated in each Island of Reil.

A nodule of considerable size, which was very sharply defined, and limited to the position of the pyramidal tract in the right half of the pons Varolii, had evidently been the cause of the paralysis,

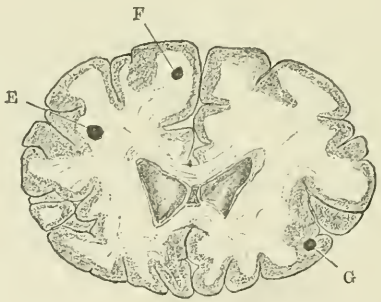


Fig. 2.—*Transverse Vertical Section through the Brain of S. E. M., at the level of the anterior end of the Corpus Callosum, showing three small Melanotic Masses* (E, F, G). (Reduced from a Photograph.)

which was at first limited to the left arm, but which ultimately involved the left leg.

The middle lobe of the cerebellum was almost entirely destroyed by a large melanotic mass, and there were several other smaller deposits in its lateral lobes. In this respect this case forms a very remarkable contrast with a case of diffused melanotic sarcoma which came under my observation while I was pathologist to the Edinburgh Royal Infirmary, and which I have very carefully studied. In that extraordinary case, which was under the care of Professor Fraser during life, and which I trust he will publish, the cerebrum, and I may add, almost every

other organ in the body, including the skin, was infiltrated with melanotic deposits. The gray matter of the cerebrum and the basal ganglia were extensively involved; but, strange to

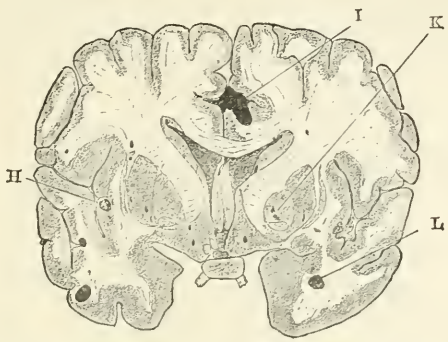


FIG. 3.—*Transverse Vertical Section through the Brain of S. E. M., at the level of the Optic Chiasma, showing numerous Melanotic Deposits, the majority of which are of small size* (Reduced from a Photograph.)

The letter H points to a small nodule in the left Island of Reil; I, to a large superficial mass in the anterior longitudinal fissure; K, to a small mass in the right lenticular nucleus; L, to a nodule in the white matter of the right temporo-sphenoidal lobe. Many smaller nodules (which are unlettered) are seen in both hemispheres. In this section the nodules on the two sides of the brain are remarkably symmetrical in distribution.

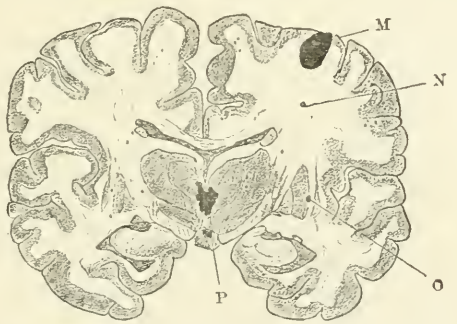


FIG. 4.—*Transverse Vertical Section through the Brain of S. E. M., just behind the Optic Chiasma, showing the position of the Melanotic Nodules.* (Reduced from a Photograph.)

The letter M points to a large nodule at the vertex; N, to a small commencing nodule in the white matter of the centrum ovale; O, to a small nodule in the right lenticular nucleus (there are several small nodules in the lenticular nucleus and external capsule of the left side); P, to a nodule of some size involving symmetrical parts of the optic thalami.

say, there were practically no deposits (or, at the most, one or two minute, almost microscopical deposits) in the cerebellum[1] (see Figs. 8 and 9).

[1] The fact that in this case no deposits were found in the gray matter of the cerebellum, while the whole of the gray matter of the other parts of the brain was infiltrated, is strongly suggestive of some radical difference in the distribution of the vessels and the arrangement of the lymphatic spaces in these two important nerve centres.

In some parts of the brain of Dr Jamieson's patient, the nodules of melanotic sarcoma in the two hemispheres were strikingly symmetrical in position (see Fig. 3),—a fact which is probably to be explained by the circumstance that the sarcomatous elements were, in all cases, grouped round, and probably had been carried from place to place by, the bloodvessels.

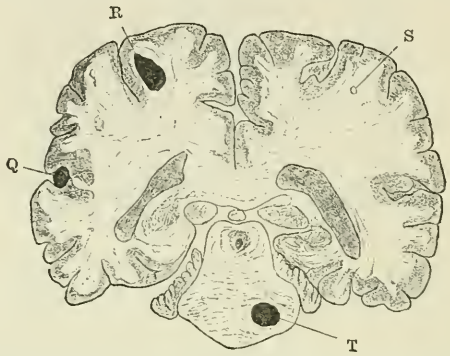


FIG. 5.—*Transverse Vertical Section through the Brain of S. E. M., at the level of the greatest convexity of the Pons Varolii, showing the position of Melanotic Nodules* (Q, R, S) *in the Brain, and a large Nodule* (T) *in the Pons, involving the right Pyramidal Tract.* (Reduced from a Photograph.)

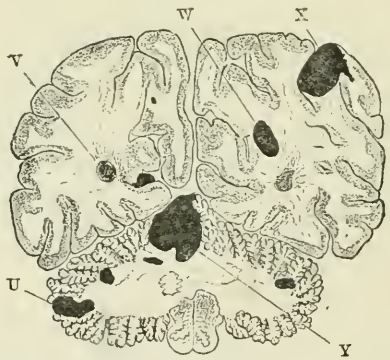


FIG. 6.—*Transverse Vertical Section through the Brain of S. E. M., at the level of the Olivary Bodies of the Medulla Oblongata, showing numerous large Melanotic Masses, both in the Cerebrum and Cerebellum.* (Reduced from a Photograph.)

The letter U points to a nodule of some size in the left lateral lobe of the cerebellum; V, to the lateral ventricle of the left side; W and X, to large nodules in the right hemisphere of the brain; Y, to a very large nodule in the middle lobe of the cerebellum.

On microscopical examination the nodules of new growth were found to be typical examples of melanotic sarcoma.

In the older growths the cells varied in size. Some were small and round (three or four times the size of a red blood corpuscle), and containing a single round nucleus about the size of a leucocyte (see Fig. 12, B and C). Others were very large, round or

angular; many of these large cells contained two, three, or even four or more nuclei (see Fig. 12, A).

The small (young) deposits in the brain were almost entirely composed of small round cells, which were clustered round dilated

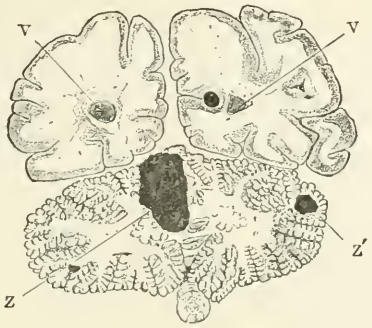


Fig. 7.—*Transverse Vertical Section through the Brain in the case of S. E. M., at the level of the lower end of the Medulla Oblongata, showing the position of the Melanotic Nodules.* (Reduced from a Photograph.)

The letters V, V, point to the lateral ventricles; Z, to a large mass of new growth involving the middle lobe of the cerebellum; Z′, to a small nodule in the right lateral lobe of the cerebellum.

bloodvessels. The vessels in the brain tissue, surrounding the deposits of new growth, were greatly dilated (see Fig. 10).

In the smaller (younger) nodules, the pigment consisted of very minute brown granules. In the larger and older nodules, many of the pigment granules contained in the sarcomatous cells were considerably larger (see Fig. 12, D, D). Free granules and masses of pigment granules were in places seen lying apparently free in the midst of the sarcomatous elements.

In the other case of melanotic sarcoma, to which I have referred, the cells were situated in the lymphatic space surrounding the bloodvessels; and, in consequence of the pigmented character of the cells and their situation, the course of the minute vessels and lymphatic spaces in the gray matter of the brain could be traced out in a most remarkable way (see Figs. 13, 14, and 15).

The connective tissue corpuscles throughout the brain, but especially in those parts which surrounded the sarcomatous nodules, were very numerous and large.

The microscopical characters of the melanotic deposits in the tonsil, lymphatic glands, and kidney were identical with those in the dura mater and cerebrum.

In one section of the kidney, a vessel, which was situated at a distance from any melanotic mass, and which was surrounded by healthy renal tissue, was filled with pigmented sarcomatous elements, which seem, so far as I can judge, to have been present during life, and to have formed a true sarcomatous infarct.

The nodules of new growth were of very soft consistence; in the recent state they were so soft as to be almost diffluent when

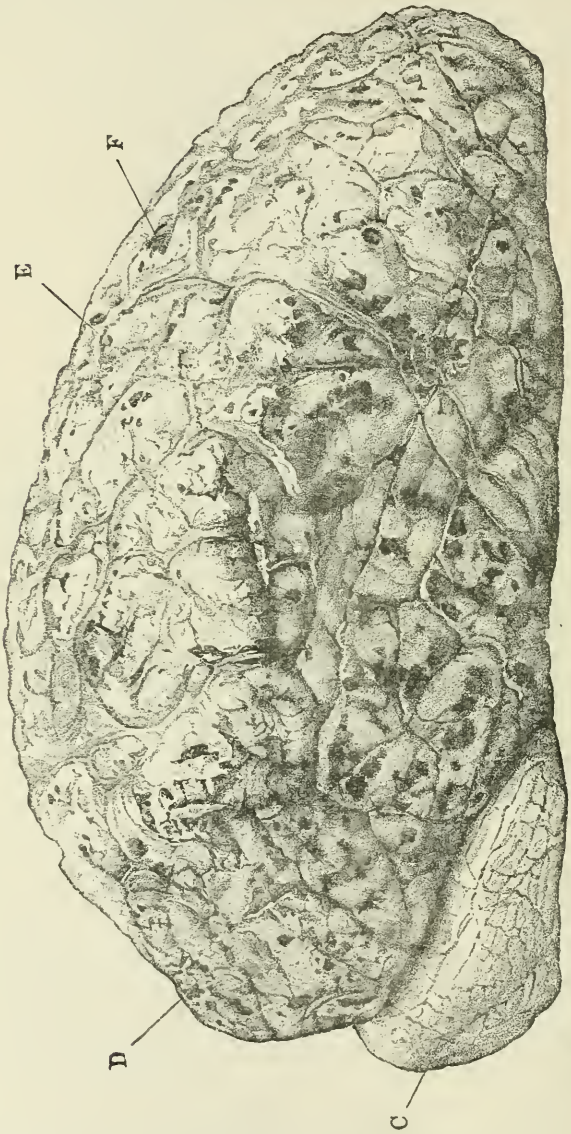


Fig. 8.—*Lateral View of the right side of the Brain and Cerebellum in case of Diffused Melanotic Sarcoma.* (Reduced from a Photograph.) The letter C points to the cerebellum, on the surface of which no melanotic deposits are to be seen; D, E, and F, to melanotic deposits on the surface of the cerebrum. The whole surface of the brain is studded with similar deposits.

cut into. Even after hardening in Müller's fluid and in spirit, the sarcomatous elements could be washed away with the greatest ease from the cerebral vessels around which they were massed.

Remarks.—The case presents several points both of clinical and

pathological interest in addition to those to which Dr Jamieson has referred.

The fact that there was no return of the sarcomatous growth at its original seat, and the circumstance that the secondary deposits in the brain and other organs did not begin to manifest themselves until after the lapse of nearly eight months after the removal of the primary source of infection, are points of importance.

The absence of any secondary deposits in the glands of the right

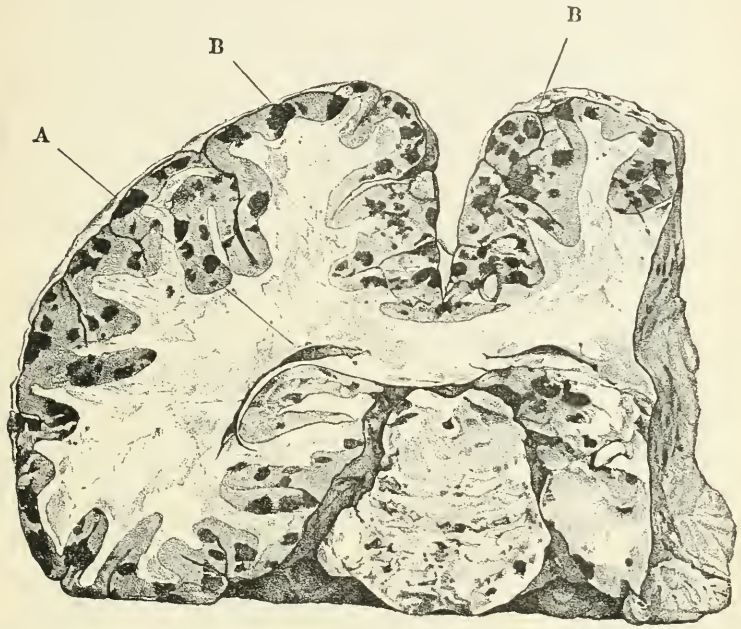


FIG. 9.—*Transverse Vertical Section through the left and portions of the right Hemisphere of the Brain, at the level of the greatest convexity of the Pons Varolii in case of Diffused Melanotic Sarcoma, showing an enormous number of Melanotic Deposits in the Gray Matter of the Cerebrum and in the Substance of the Pons Varolii.* (Reduced from a Photograph.)

The letter A points to a spot just above the lateral ventricle on the left side; B and B, to melanotic deposits in the gray matter of the cerebral cortex.

side of the neck, within the thorax, or in the liver, spleen, and skin, is also noteworthy. The relationship of the sarcomatous cells to the bloodvessels and lymphatic spaces is of much interest, and has been already referred to.

The symmetrical distribution of many of the sarcomatous deposits in the two hemispheres of the brain is a point which has been already emphasized.

The practical absence of any deposits in the motor region of the cerebrum or in the internal capsules; and the circumstance that a paralysis, which was for some time limited to the arm (though it subsequently also involved the leg), was produced by a localized

lesion in the pons Varolii, is noteworthy, for it very rarely happens that a lesion of the pons, whether acute or chronic, produces a paralysis of one limb only (monoplegia).

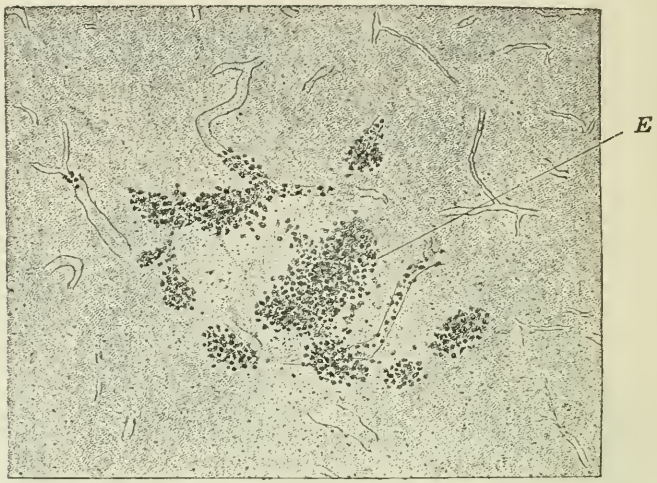


FIG. 10.—*Microscopical Section through a portion of the White Matter of the Brain in the case of S. E. M., shewing a Nodule of Melanotic Sarcoma in an early stage. Lower power (Hartnack, ocular 3, objective 4, and tube out; Drawing reduced from 3⅞ to 3⅜ inches).*

The sarcomatous cells are massed round the bloodvessels; some of the vessels in the surrounding portions of the brain substance are very much dilated.
The letter E points to a mass of sarcomatous cells.

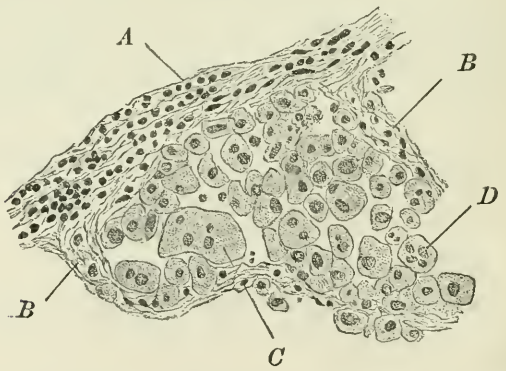


FIG. 11.—*Microscopical Section through a Nodule of Melanotic Sarcoma attached to the Dura Mater in the case of S. E. M. (Magnified—Hartnack, ocular 3, objective 8, and tube drawn out.)*

The letter A points to the deepest layer of the dura (a small portion of which is only represented, for the membrane was very much thickened). The dura is seen to be infiltrated with deeply stained leucocytes and enlarged connective tissue corpuscles. The letters B, B, point to septa of fibrous tissue (which are also infiltrated with leucocytes) passing from the dura into the tumour tissue; C and D, to large sarcomatous cells containing several nuclei.

The absence of giddiness, notwithstanding the fact that the

middle lobe of the cerebellum was so extensively invaded, is a point of importance. This is all the more noteworthy, since the

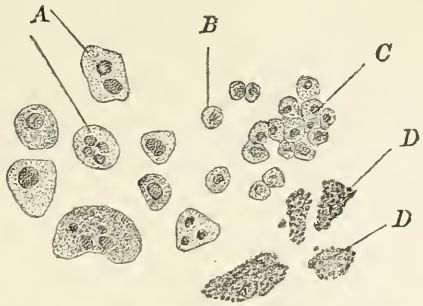


FIG. 12.—*Individual Cells from Sarcomatous Nodules in the case of S. E. M. (Magnified—Hartnack, ocular* 3, *objective* 8, *and tube drawn out.)*

The letter A points to large cells from a nodule in the dura mater; B and C, to small round cells from a commencing nodule in the brain; D, D, to large cells filled with large melanotic granules from a nodule in the tonsil.

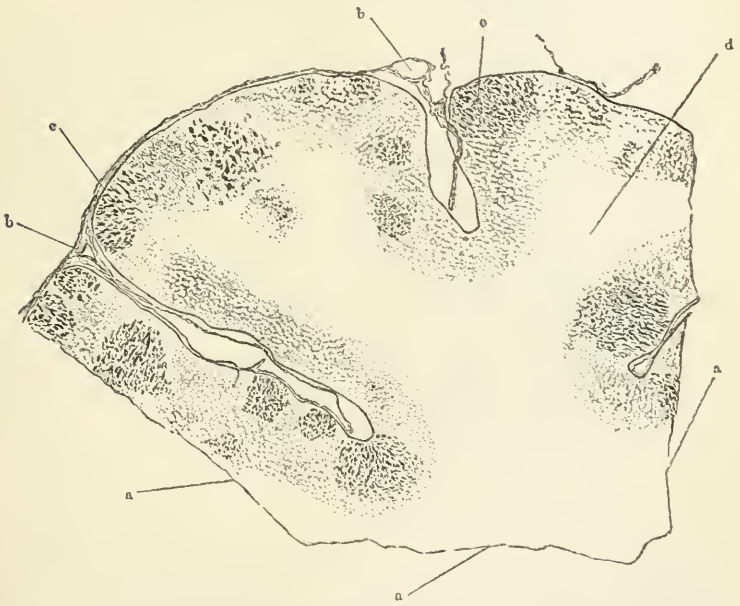


FIG. 13.—*Camera Lucida Drawing of a Microscopical Section through a portion of three Convolutions of the Brain in case of Diffused Melanotic Sarcoma, showing numerous Sarcomatous Nodules entirely confined to the Gray Matter. Very low power. (Hartnack, ocular* 2, *objective* 1, *and Drawing reduced from* 8 *to* 4 *inches.)*

The letters *a*, *a*, *a*, point to the cut edges of the section; *b*, *b*, to transversely divided vessels in the membranes on the surface of the brain; *c*, *c*, to masses of melanotic sarcoma in the gray matter; *d*, to the white substance in the centre of a convolution of the brain.

effect which the lesion produced on the nervous elements must,

I think in the earlier stages at all events, have been essentially irritative.

FIG. 14.—*Camera Lucida Drawing of a Microscopical Section of a portion of the Gray Matter of the Cortex of the Brain in case of Diffused Melanotic Sarcoma, showing a Melanotic Mass under a lower power. (Hartnack, ocular 3, objective 4, tube drawn out, and Drawing reduced from 7 to 5½ inches.)* The pigmented sarcomatous cells (the individual outlines of which it is difficult to distinguish under this magnifying power) are grouped in a most remarkable manner around the bloodvessels.

The letter A points to the membranes on the free surface of the convolution; B, to a portion of the gray matter which is not invaded; C, C, to masses of pigmented cells surrounding the vessels.

The very marked paroxysmal character of the headache, and

the very striking vaso-motor disturbance (flushing of the face, alteration in the pulse, and sweating) which accompanied the

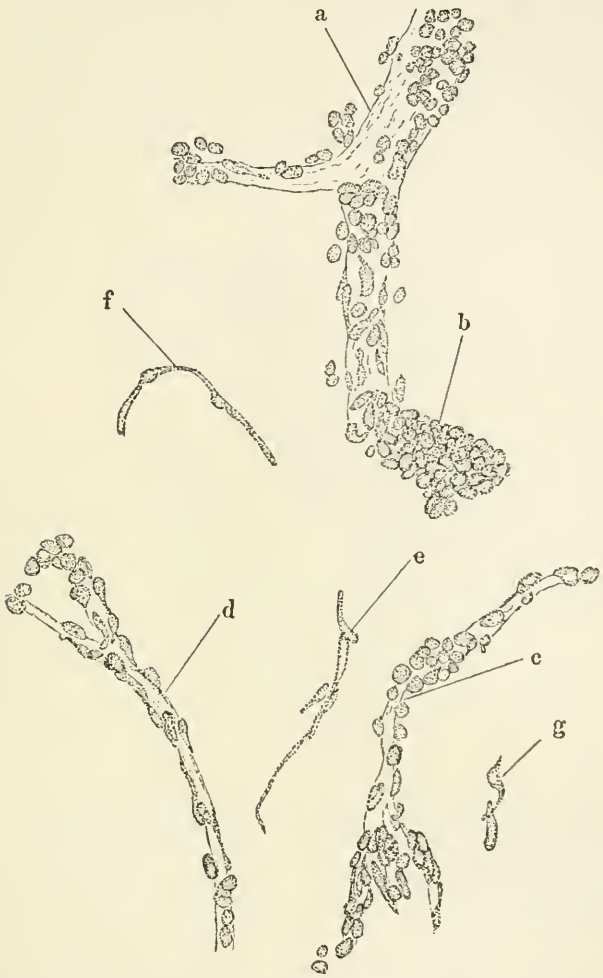


FIG. 15.—*Camera Lucida Drawing of Bloodvessels from the Gray Matter of the Brain in case of Diffused Melanotic Sarcoma, showing Vessels and Pigmented Sarcomatous Cells surrounding them. High power.* (*Hartnack, ocular* 3, *objective* 8, *tube out, and Drawing reduced from* 6½ *to* 5 *inches.*)

The letter *a* points to a vessel of some size at a point where the vessel wall is unsurrounded by cells; *b*, to a large mass of cells surrounding a vessel; *c* and *d*, to small vessels surrounded with cells; *e*, *f*, and *g*, to pigmented sarcomatous cells of various shapes, which are making their way along narrow lymphatic spaces, and are in consequence very finely drawn out.

headache are of the greatest interest; indeed, I know of no case of cerebral tumour in which such very marked vaso-motor alterations have been observed. The exact position of the lesion which

produced this vaso-motor disturbance would be a point of great importance, but I confess that in the presence of so many secondary nodules in the cerebral and cerebellar tissues, I do not like to hazard an opinion on this point.

Another symptom of great interest was the electric-like flashes of light which, according to the patient, were more marked in the right than in the left eye.

There can, I think, be little doubt but these flashes of light were

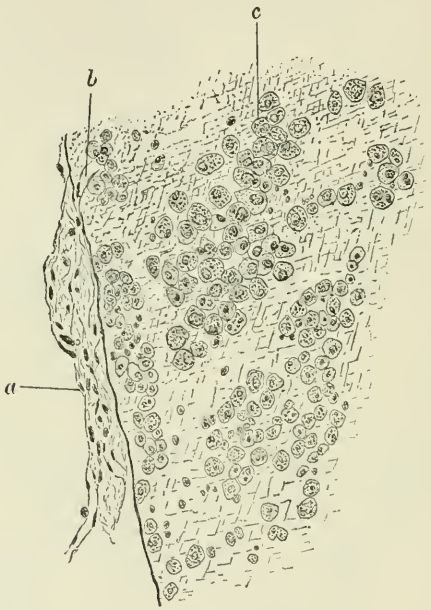


Fig. 16.—*Camera Lucida Drawing of a Section through the surface of a Convolution of the Brain in case of Diffused Melanotic Sarcoma. High power. (Hartnack, ocular 3, objective 8, tube out, and Drawing reduced from 2½ to 2 inches.)*

The letter *a* points to the membranes on the free surface of the convolution; *b*, to a small (capillary) vessel passing into the gray matter; *c*, to a group of large, rounded, and, in some cases, multi-nucleated sarcomatous cells containing pigment.

epileptiform in character (sensory epileptiform attacks), and that they depended upon a discharging lesion of some part of the cortical centres for vision. The fact that they were said to be more marked in the right than in the left eye is highly suggestive, that the discharge upon which they depended was due to a lesion of the posterior part of the left cerebral hemisphere (region of "visual centre" on the *left* side), for it must be remembered that flashes of light which are due to a central discharge, and which are said by the patient to be seen in one eye or on one side, in most cases, at all events, depend upon a discharge of nervous elements corresponding *not* to the whole of one (the opposite) retina, but *to one-*

half of each retina. In this case, in which the flash of light was seen on the right side, and chiefly referred to the right eye, the nervous elements corresponding to the inner (nasal) side of the right and the outer (temporal) side of the left retina were, I take it, discharged. The presence of a large secondary melanotic deposit on the surface of the left occipital lobe, just above its tip, is strongly corroborative of this theoretical view; and there can, I think, be little reasonable doubt that the flashes of light were the result of that particular nodule.

The bright electric-like character of the flashes of light deserves notice, for in the great majority of cases of epilepsy which have come under my observation, in which a flash of light was observed, the colour of the light has been red. In some cases I have, however, known the red accompanied by white, or gradually fade into white or blue, as the discharge progressed and disappeared.

The Chairman remarked on two points of interest in the case. The first was the hæmorrhagic mark on the mother's thigh, caused by a playful grip of her husband's hand, and whether this had anything to do with the tendency to the occurrence of the pigmentary tumours in her daughter in after life it was impossible to say, but the coincidence was curious. The second was, that while these tumours were usually supposed to occur in dark people, this girl was reported to be fair.

Dr Woodhead supposed those melanotic sarcomata gave more trouble and thought to the pathologist than almost any other form of sarcoma. They had in the two cases before them, as given by the authors of the paper, examples of two distinct methods of invasion, in one instance spreading occurring by lymphatics, in the other by bloodvessels. It was very difficult to determine how they had those tumours, so like in many respects, spreading apparently in quite a different manner. One important point in this paper was the connexion Drs Jamieson and Bramwell made out between the primary hæmorrhage with irritation and the secondary tumour. Lately, Dr W. had examined a number of these tumours, and it always struck him that, apart from other nutritive and constitutional changes, there was a distinct relation between the growth of melanotic tumours and a primary and secondary irritation. By a primary irritation he meant some such irritation as a blow, or continuous irritation such as rubbing on the shoulder, as in the case mentioned, but there was also a secondary irritation from the blood pigment, which acted as a foreign body on the connective tissue cells in the neighbourhood of this primary irritation, and there appeared to be more due to this blood pigment acting as a foreign body on the cells lining the lymphatics than they were ordinarily inclined to credit. This

pigment detached from blood corpuscles was as much a foreign body as dust. It was a constant source of irritation to the cells in the lymphatics, and one could conceive that they had a continuous irritation and proliferation of cells in the lymphatics. They had this pigment passed on from cell to cell, the proliferation continuing, and they had a sarcoma. But as this grew, as the area occupied by it increased, they had the embryonic blood-vessels formed and secondary hæmorrhages. This had been broached by several writers, and it seemed to him one of the most rational explanations of a very difficult question. The case described appeared to throw some considerable light on these tumours, but he should still like more light as to the method of their distribution. Dr Bramwell pointed out that the paths were perfectly distinct, though they had the same result, the same structures, and same pigmentation in both.

Dr Foulis drew attention to the fact that these tumours occurred in the skin, and secondarily in the brain. These tissues were derivatives of the same embryonic layer, viz., the epiblast.

Dr Woodhead asked if it were not the case that organs derived from the mesoblast and hypoblast, such as the liver, mesenteric glands, etc., were equally affected with those derived from the hypoblast?

Mr Cathcart remembered a case of melanotic sarcoma under the care of Dr MacGillivray, in which the disease began primarily in the glands of the groin. He had also assisted Mr Bell in the removal of a large sarcoma from the orbit of a girl, in whom the growth had very rapidly followed a blow on the eye from a snow-ball. Both of these patients were fair. In both there was rapid recurrence.[1]

Dr Wm. Russell remarked on the tendency there had been in the discussion to accentuate the influence hæmorrhage might have in the production of melanotic tumours. He was not prepared to discuss the origin of these tumours fully, but he should like to draw attention to the observations of Sir James Paget, made many years ago, in which he contended there were two conditions necessary to give rise to this type of tumour. He insisted, first, on a certain constitutional condition being present at the time which he called specific (not syphilitic), but the exact nature of which he did not attempt to define. The second condition was that of local injury. He pointed out that the same kind of injury might be repeated many times in the same case, and that after the earlier injuries they had no sarcomatous growths, but that after the later injuries they might have the development of a melanotic sarcoma. In this way it was necessary that they should have this specific constitutional condition. Sir James illustrated his contention by cases resembling those mentioned. The point

[1] See *Edinburgh Medical Journal*, March 1885.

that Dr Woodhead brought forward with regard to the influence of melanin in the production of these tumours was one of great interest, and he had no doubt that with the increased opportunities for investigation now at his disposal he would be able to satisfy himself whether melanin was really capable of irritating the animal tissues in this way. Blood pigment, they had abundant proof, did not act as an irritant. That these tumours usually followed on pigmentary moles which had been injured or irritated in some way was of great importance. Their mode of secondary distribution was also of interest. That in the majority of cases they undoubtedly spread by lymphatics, no one, he presumed, was prepared to question.

Dr Wm. Taylor was hardly satisfied that the affection of the brain was a *secondary* condition. He was rather disposed to think that all disease originated in the brain. When they looked at the diffusion of this melanotic material through the brain, in many parts it was a very difficult thing to believe that it arose from the little mole on the shoulder, whereas it was easy to believe that, given this pre-existing condition in the brain, a blow might set up the mischief in any part of the body. He did not think that the time given for the cerebral invasion was sufficient to allow of the extensive involvement of the cerebral tissue. A familiar instance of disease originating in the brain was found in the case of consumptives, in whom the disease undoubtedly originated there, because there was no class of patients who were so foolish about taking care of themselves, indicating a pre-existing emaciation of the brain and a secondary emaciation of the other tissues involved.

Dr James said that seeing the discussion had become a little heterodox, he might be allowed to suggest that tumours should be looked upon as conditions that arose when the nutrition of a part was weakened. Given an individual, any of whose tissues were below par, he would be liable to the formation of tumours in those weakened parts. The exciting cause might be an injury or some long-continued irritation. Given a deficient nutrition, he would like to suggest whether in the cases where the brain was affected it was not legitimate to suppose that the nutrition of that organ was below par, and so the affection developed there rather than spread elsewhere by the lymphatics.

Dr Wm. Taylor thought the result of a blow or some continuous irritation on a weakened part would depend on the pre-existing condition of the brain. In one individual the result would be a callosity, in another a wart, and in a third a sarcoma would be produced.

Dr Byrom Bramwell, in reply, said that while he was by no means disposed to undervalue the effect which conditions of mind had upon the nutritive changes and nutrition of the body, he saw no reason for accepting the suggestion which had been advanced by Dr Taylor, and to some extent endorsed by Dr James, as to the

mode of production of the melanotic deposits in the brain in this particular case. A localized pigmented mole had after injury taken on sarcomatous action; the new growth had been removed. After an interval of some years the melanotic sarcoma had returned, and again had been removed. Then, after an interval of several months, cerebral symptoms had developed, and after death numerous and, for the most part, very small and therefore young sarcomatous nodules were found scattered throughout the brain tissue. The sarcomatous elements were massed round the bloodvessels. Dr Bramwell could not doubt from these facts that the sarcomatous nodules in the brain were secondary, and had in all probability been distributed through the blood-vessels. The relationship of the lymphatic channels to the vessels of the brain make it extremely difficult to determine in some cases whether a new growth which is grouped round blood-vessels has been carried through the bloodvessels or through the lymphatic channels. Dr Foulis's question had, he thought, been sufficiently answered by Dr Woodhead. In four cases of melanotic sarcoma which he had seen, the sarcomatous nodules had been pretty generally distributed throughout the tissues. The exact causation of melanotic tumours was a very difficult question. The view which Dr Woodhead had advanced was ingenious. It was too difficult to disprove by mere microscopical examination another view which had been propounded, viz., that the pigment molecules were living organisms. Personally he was disposed to think that the sarcomatous cells themselves were endowed with the peculiar property of separating from the blood—manufacturing from the blood, so to speak—the pigmentary material. The pigment was present in the form of fine granules in the interior of the cells themselves, and, in some instances, in the form of larger granules massed together, apparently free, between the cells.

2. ON THE REMEDIES USED BY THE CAFFRES TO PREVENT BLOOD POISONING FROM ANTHRAX.

By R. Peel Ritchie, M.D., F.R.C.P. Ed., etc.

It is now upwards of three years since my friend Mr Frederick Wienand of Bellevue, Bedford, Cape Colony, forwarded to me the two specimens of plants I submit to the Society, but various circumstances delayed me making use of them. The *first* was the difficulty I experienced in getting the plants identified. The *second*, my unsuccessful efforts to learn further particulars regarding their use, or that of other plants or remedies for a similar purpose in other parts of South Africa. None of the books I happened to look into afforded me any information. Mr H. M. Stanley, the African traveller, with whom I communicated

when he was last in Edinburgh, could throw no light upon the subject, and to Mr Joseph Thomson it was also unknown. During the time the Forestry Exhibition was open in this city, I tried to learn if the representatives at the South African Court could give me information or identify the plants, but I was again disappointed. Nor was I more successful in my application to the officials at the Botanic Gardens, nor amongst the practical horticulturalists with whom I was acquainted. I then took counsel with a friend in London, who, although himself a good botanist, preferred to submit the specimens at the Botanical Department of the British Museum. In doing so, he unfortunately said they were from South America, and after much unnecessary searching, the report was, they could not find any plant with which they could associate them. Determined not to be left in this very unsatisfactory position, application was again made, and the correct source of the specimens given, and I was ultimately successful in getting them identified.

Since I succeeded in obtaining the names of the plants, I have endeavoured to learn if there was any record of the use of plants by the natives for the like purpose, but I have failed to find any reference. Amongst British natives with whom I have spoken, probably from their having come from other parts of the country, and possibly from their not having been engaged in farming, there was knowledge neither of the disease nor of its prevention. The veterinary surgeons in this city, from whom I made inquiry, knew nothing of the use of drugs for the purpose, and there is only one authority I know of (from whose book I shall shortly quote), who mentions the eating of the flesh of cattle affected with splenic fever or anthrax. At this stage of the inquiry a new difficulty occurred. On questioning two fleshers engaged at the slaughter-house here in slaughtering and dressing animals, after describing the pathological appearances in cattle dying of splenic fever or anthrax, they said they were quite familiar with them, but they called the disease and knew it amongst themselves as the "black spauld or spaud," and that they had often eaten the flesh of animals dying from it and dressed by themselves. Black spauld being a Scottish term for "quarter ill," or "emphysema infectiosum," did not occur in the books on veterinary medicine I was able to consult, and it is only lately, through the kindness of Principals Williams and Walley, that I have learned full particulars regarding it, and satisfied myself of the distinguishing characteristics between anthrax and "black spauld." From the observations of the practical fleshers I have referred to, I have a fear that animals suffering from anthrax have not only been slaughtered, but that the flesh has also been eaten. On this point, however, Principal Walley informs me "the Edinburgh fleshers know nothing of anthrax. It is most dangerous in every way. We have no proof that quarter ill is ever transmitted to man, and

the sound parts of carcase are used as human food with impunity; the infected fluids do not even kill cattle if introduced directly into the blood." I think it right to state that I am informed by the authorities at the slaughter-house, that the flesh of cattle affected with quarter ill is condemned, and that the carcase affected with anthrax is destroyed by fire.

My conversation with the fleshers has also delayed me in submitting the specimens to the Society, for, if these men have killed, dressed, and eaten anthrax animals with impunity, and without the previous use of an antidotal remedy, then possibly the South African Caffres might do the same?

The recent allusions to anthrax in the House of Commons, and the ignorance regarding it as shown by the occurrence of cases in Cheshire, in "repeated outbreaks," make this a suitable time to bring the subject before you.

When Mr Wienand visited me, in course of conversation the subject of splenic apoplexy came up. He stated that it was very common in some parts of South Africa, especially so in his neighbourhood, and caused the rapid death of many oxen. He also alleged that the native Caffres feasted to gorging on the carcases after a certain form of protecting themselves from the virus had been gone through. Mr W. promised on his return to Cape Colony to try and obtain specimens of the plants the natives used, and to send them to me. The quantity sent was, however, too small for experiment had the opportunity offered, at least until the specimens had been authenticated.

The following are Mr Wienand's observations accompanying the specimens. He writes from Bedford, Cape Colony:—

"By a friend I send you two different herbs, which the natives use in cases of blood poisoning from animals that die from splenic affection. I have obtained them at great trouble and expense, as the natives are very loth to give information on these subjects, and their cure is really a marvellous one. The two are to be kept separately, each being a distinct cure in itself. The one without the yellow flower (*Teucrium Capense*) is a good antidote for snake bites as well.

"The herbs are bruised when green, for they are always to be had, and infused in cold water, of which copious draughts are taken. To my knowledge, it has never been tried to cure cattle, there is no time for it. An apparently healthy animal drops down dead and is shunned by everybody, and if dogs feed on it they also die; but if a *native specialist* is comeatible he is sent for; a throng in the meantime approaches the carcase, and the doctor administers a draught to each individual, after which the animal is skinned, boiled, and devoured amidst great rejoicings. Blood poisoning only occurs when in ignorance such an animal is skinned or the meat handled."

The plants which I now show belong to different Natural Orders,

—the one to the Labiatæ, said by Mr Wienand to be also a good antidote for snake bites, has been identified at the British Museum as the *Teucrium Capense* (Thunberg); the other is a Composite plant, named by the same authority as the *Matricaria vigellæfolia*, D. C. Before receiving this information, it seemed to me to have the characters of an Artemisia, but these two genera are not very remote.

Of our British species of Matricaria, two at least are recognised to have pharmaceutic properties, namely, the *Matricaria Parthenium*, the feverfew, and the *Matricaria Chamomilla*, the wild chamomile, which is sometimes substituted for the *Anthemis nobilis* in the preparation of oil of chamomile.

Of the Labiate plant, the Teucrium, as possessing medicinal properties, there is no great record. The best known British species, the *Teucrium Scorodonia*, the wood sage, and *T. Chamædrys*, have aromatic and bitter tonic properties.

Regarding the properties of the plants I show, nothing is known at the British Museum Botanical Department.

The points I desire to direct attention to in Mr Wienand's statement are—

1*st*. That until protected by the infusion of the herbs, the natives shun the carcase of the anthrax affected animal.

2*nd*. If dogs feed on the raw flesh they die.

3*rd*. That blood poisoning occurs only when in ignorance such an animal is skinned or the meat handled without precautions.

4*th*. But after copious draughts of the infusions in cold water have been taken, the carcase is approached with freedom, and skinned and cut up.

5*th*. That after being boiled it is devoured with great rejoicings.

6*th*. That my correspondent is not aware of the diseased animal being dosed with the drugs during life, because there is no time for it.

Upon these points the following observations occur to me, but I premise them by this statement regarding the transmitter of the information.

Mr Wienand is a native of Cape Colony. He is an extensive landowner, and possesses a large amount of stock in cattle, sheep, and ostriches. He is a very intelligent man, not likely to be readily misled in his judgment, his mind being enlarged by travel, and he has, moreover, a thorough knowledge of the native Caffres. The information he supplies is likely to be reliable therefore. As evidence of his intelligence and stock-rearing ability, it may be added that in one year (1883), at the Agricultural Exhibition, he "was very successful, taking 17 prizes, 14 of which were first class for stock, heading the list of competitors by a long way."

Of the danger attending contact with the dead animal the natives are well aware. That this is great, the statement that unprotected men and dogs are affected proves that it is so. Dogs eating

the uncooked flesh die. It is important to note this, for if the operator on the dead animal is protected by draughts of the infusions, he can cut up the animal apparently with impunity. It is upon this statement that the evidence of protection depends, for after the animal is cut up *and cooked*, we know from home evidence the meat may be eaten without injurious result.

The fact that the treatment has not been applied to the diseased animal does not, it seems to me, have much bearing on the present inquiry. If there had been evidence that after administration the animal survived, the proof would not have been great, for it might be alleged, readily and feasibly, that the animal had not suffered from splenic fever or anthrax. This evidence would be therefore of doubtful import.

The real point of importance is that without protection there is great danger, and after taking the remedy there is apparently none.

Cooking the flesh of an anthrax affected animal destroys the vitality of its bacillus. Once it is in the pot, the meat may be eaten after cooking with impunity. This I learned from the fleshers; and since my attention has been directed to the subject, my friend Mr Alex. Inglis M'Callum, V.S., favoured me with the perusal of the excellent work by Principal William Robertson, Professor of Hippopathology in the Royal Veterinary College, London, *A Text-Book of the Practice of Equine Medicine*, 1883. He gives a full and excellent account of anthrax, its history, literature, geographical distribution, and pathology.

I quote the following from his book (page 183) as to the introduction of the disease by ingestion:—"Deductions derived from experiment and the behaviour of the bacillus anthracis in relation to artificially produced disease, compel us to subscribe to the theory of its being communicable by the alimentary tract, the air-passages, or by cutaneous inoculation, and, though generally fixed, the virus may be more rarely volatile, that it is transmissible is now beyond dispute." He further remarks: "To the condition of receptivity of the species and individual we are inclined to attach the utmost importance, and from our observation we are led to look to this for much explanation as to the apparently erratic behaviour of the anthrax virus."

After referring to the effects of temperature on the vitality of the filaments and spores, he states at page 195,—"I have myself partaken daily for some days of roasted beef from an ox killed because affected with splenic fever, without unpleasant results." "Boiling for two minutes entirely destroys rods and spores in all conditions—fresh or old, dry or moist;" and at page 198 his fifth deduction is "that the boiling point is fatal to the spores and rods."

The mere fact that these Caffres can eat and enjoy one of their voracious meals, after cooking the flesh of an anthrax animal, is of small importance after learning Mr Robertson's personal and

experimental experience, but I submit a case for further inquiry as to the effect of protective means has been made out. Although we do not in this country recognise any special therapeutic quality in the indigenous species of Matricaria and Teucrium, still it may be kept in remembrance that, in addition to their stomachic and tonic properties, chamomiles, though inferior to many other medicines, in the words of Pareira, "as a remedy for intermittents . . . have gained considerable celebrity."

I should not wish to be understood as advocating the use of the flesh of anthrax infected cattle. My intention has been to raise inquiry as to the protective means to be used by those who unfortunately have to handle the diseased carcases and hides; and the subject is one to which the attention of the Colonial Office should be called, so that, in conjunction with the authorities at the Cape, full inquiry may be made in the districts where this protective means is used.

Before I resume my seat may I add this deduction, although not strictly connected with the subject of my paper, that heat is the best destroyer, not only of the rods, but of the germs of the anthrax bacillus. Experience recorded so far back as the year 1873 (in the *Veterinarian* for December), has also proved the danger which may follow the burying of these diseased animals if in such a position that the water-drainage can affect the pond from which the animals on a farm receive their water supply. We now know the explanation of this, yet it is remarkable that with fourteen years' interval burying is still permitted, and explanations sought for the prevalence of the disease! Boiling or cremation are undoubtedly, to my mind, the best methods of disposing of the diseased carcase. Whichever is practicable ought to be employed.

Dr Woodhead said one or two points had specially struck him as Dr Ritchie read his paper. The first was that the Caffres did not touch the animal before the medicine man came up. This was a very important point, because so long as no wound was made and the carcase was left unopened the spores of the anthrax bacillus did not develop. If the boiling took place immediately after the animal was cut up, there was no time for the development of the spores, and there was thus little danger of anthrax-poisoning, because a comparatively low temperature killed the bacilli. Another point was whether this was anthrax at all. Dogs were not usually susceptible to anthrax, nor were other animals when the bacilli were introduced by the intestinal canal. Dr Woodhead had fed a dozen rats with the liver and spleen of a cow that suffered from anthrax, and only two of these were affected through the intestinal canal. Even mice were not so markedly affected in this way, though eighteen hours were sufficient to kill one after an inoculation. He should like to try if an infusion of these plants would prevent the growth of spores in a gelatine cultivation.

Dr Bramwell suggested that Dr Ritchie should hand over to Dr Woodhead some of the plants for experimental investigation in the new laboratory of the College of Physicians.

The Chairman drew attention to a curious coincidence. Both anthrax poison and snake poison could be swallowed with impunity, and it was worthy of note that one of these plants was said to be a remedy for or preventative of snake poison, though this might be when the poison entered by a bite.

Dr Peel Ritchie, in reply, said that his friend having sent him these plants, his duty after their identification was to bring them before some scientific society. His own feeling was one of doubt. The receptivity of the individual undoubtedly varied, and it was probably by altering this receptivity that these drugs seemed to act. The point Dr Woodhead had suggested was possibly the correct explanation, for it was shown that the meat from an anthrax infected animal might after cooking be eaten with impunity. The only difference between Mr Robertson and Dr Woodhead was that Robertson on several days ate meat from an animal that suffered from anthrax under conditions that would allow of the development of spores. He would be glad to furnish Dr Woodhead with material for investigation, as his objects in bringing the subject before the Society were to elicit opinion and court inquiry.

Meeting VIII.—June 1, 1887.

Professor GRAINGER STEWART, *President, in the Chair.*

I. ELECTION OF ORDINARY MEMBER.

Edward Carmichael, M.D., Leith, was elected an Ordinary Member of the Society.

II. EXHIBITION OF PATIENTS.

1. *Dr Argyll Robertson* showed a case of ENLARGEMENT AND DISPLACEMENT OF THE LACHRYMAL GLAND INTO THE UPPER EYELID. The case was unique in his experience, and also, he believed, in ophthalmic literature. There were cases of enlargement into the orbit which tended to displace the globe. The displacement of the gland in this case was into the upper lid, in which it formed a tumour, occupying its whole length and part of the breadth, preventing its movements. From the history, that it commenced with a degree of uneasiness and increased lachrymation, and from its granular feel and the circumstance that though attached above it was freely movable under the skin, Dr Robertson came

to the conclusion that the tumour was the gland displaced, and undertook its removal. During the operation he came across the accessory portion of the gland in its normal position, and left it there to provide moisture for the eye, which had quite recovered from the operation. The lid did not move so readily as its fellow, but its movements were much better than before. The patient had deep sunk eyes and abnormally small orbits, which afforded a probable explanation of the displacement into the lid instead of the orbit. The patient was a spirit merchant, and had sustained no injury to the eye, nor had he any inflammatory symptoms. The tumour was observed by the patient in January, when it was about the size of a bean, since which time it had gradually enlarged. The patient also stated that in the winter of 1885, a small similar swelling occurred in the lid, which, however, did not inconvenience him much, and disappeared. The removed gland was also shown.

2. *Mr A. G. Miller* showed TWO PATIENTS who had suffered from CEREBRAL ABSCESS connected with ear disease. The first was admitted about a year ago to the Royal Infirmary, for what was apparently a mastoid sinus. Dr M'Bride saw him, and agreed that trephining of the mastoid cells was necessary. On making the incision an opening was found in the bone which was connected with a cavity from which cheesy matter was removed, and the walls of which were not osseous. A good recovery was made, healing taking place within three months. He was readmitted a short time ago with a return of the abscess, which was opened and again scraped. The abscess caused no brain symptoms of any kind. The sinus was $1\frac{1}{2}$ inches above the tip of the mastoid, and an examination of a number of skulls showed that this corresponded with the junction of the squamous and petrous bones. A probe passed through the sinus $1\frac{1}{2}$ inches forwards and inwards, and a comparison of skulls indicated that the point thus reached was the roof of the tympanum. The second case was sent by Dr M'Bride. The patient had perforation of the membrana tympani, with discharge, and an abscess behind the ear. Dr M'Bride thought the mastoid cells were not affected, and the abscess turned out to be superficial. He continued, however, to complain of great pain over the eye and side of the head. A soft puffy swelling appeared above the ear. This turned out to be an abscess, which was thought at first to be specific, as there was a specific history. After opening it a sinus was found leading through the skull to the dura mater, situated about $1\frac{1}{2}$ inches above the ear. This healed, leaving a depressed cicatrix. Dr M'Bride was of opinion that this was a case of suppuration taking place in connexion with the middle ear. To explain the somewhat remarkable course of the pus in these two cases, Mr Miller made a careful examination of eight skulls, and found in most of them that there was a slight de-

ficiency in the closure of the suture of the parietal and petrous portions of the temporal bone. This potential aperture was situated exactly where the sinus was in the first case, and almost in the situation suggested by Dr M'Bride for opening the skull in cases of intracranial suppuration arising from disease of the middle ear. In regard to the second case, Mr Miller thought that the pus, unable to find an easy exit, such as occurred in the first case, had passed upwards and outwards through the oblique slit that lies between the parietal and temporal bones. A considerable obliquity in the sinus in this case gave countenance to this view.

3. *Dr Byrom Bramwell* showed TWO CASES OF WORD BLINDNESS. In the first case the point of greatest interest is the fact that the patient, who was absolutely ignorant of the individual letters, and did not even recognise them when they were named to him, was nevertheless able to read many words (*i.e.*, letters when taken in combination). I am not aware that such a condition has been previously described. It is usually supposed that the highest degree of word-blindness is manifested by inability to read individual letters rather than individual words—that, in short, individual letters which are generally considered to be the most elementary parts (least highly specialised) of written speech, as they undoubtedly are the first to be acquired, are the last to be lost and destroyed. It might, therefore, at first sight be argued, that the fact that a "word-blind" patient who was utterly ignorant of the individual letters as letters, was nevertheless able to read combinations of letters or words, was opposed to the great doctrines of evolution and dissolution which Herbert Spencer, Hughlings Jackson, and others have so satisfactorily applied to the elucidation of the pathology of the nervous system, and which so greatly aid the intelligent comprehension of the derangements of the functions of the brain. When, however, the matter is looked at more closely, this apparent exception will, I think, be found to conform to the general rule. For, after the preliminary difficulties of learning to read have been overcome, and the act of reading has become facile and has been frequently repeated, the individual letters are by some persons, at all events, ignored, and it is the combination of letters—syllables and individual words—to which attention is chiefly directed. In short, it may be supposed that in some persons the syllable and individual word become, after a time, less highly specialised (*i.e.*, more elementary) parts of written speech (when read) than the individual letters. If this be so, we should expect the form of word-blindness which I am now describing to be more frequent in well-educated and intelligent people, to whom the act of reading has, as it were, become automatic, than in uneducated persons, and those who before their illness could only read with difficulty, and by spelling

out (by the help of individual letters) the separate syllables and words.

The notes of the cases are as follows:—

J. F., a very intelligent man, æt. 60, who had for many years occupied a responsible position as clerk of works, was kindly sent to me on 9th March 1887, by Dr T. G. Balfour, suffering from paralysis of the right arm and aphasia.

He stated that he had enjoyed excellent health until 15th April 1886, when he lost the power of moving the thumb and forefinger of the right hand. The paralysis was only slight, and passed off in the course of a few days. Speech was not affected on that occasion.

He remained well until 7th October 1886, when he had a second attack. On this occasion, all the fingers of the right hand were powerless, but the movements of the elbow and shoulder were not affected; nor was speech implicated. He slowly and gradually recovered from this, as he did from the first attack, and continued to follow his employment as clerk of works until 22nd December 1886, when the present attack came on.

On the morning of 22nd December he felt sick when he got out of bed; loss of power in the right hand and arm, and inability to speak then developed, and were complete in the course of a few hours. Neither on this, nor on either of the two previous occasions, was the onset of the paralysis attended with coma.

At first, the loss of power was almost complete in the upper arm and forearm, as well as in the hand, but in the course of a few weeks he began to move the arm at the shoulder and elbow. There has been no perceptible improvement in the paralysis of the hand nor in the power of speaking. The leg and face were in no way affected.

On 9th March, when the patient first came under my observation, there was absolute inability to move the fingers and thumb of the right hand; the patient could pronate, but was unable to supinate, flex, or extend the hand on the wrist; all the movements of the elbow and shoulder could be performed. The muscles acting upon the elbow and shoulders were, however, still markedly paralyzed, the movements which resulted from their contraction being very feeble and unsteady.

The fingers and thumb of the right hand were rigid and bent into the palm. The right hand and forearm were swollen; and the skin felt sodden, and was more moist than that on the opposite side.

No distinct increase of the tendon reactions, as compared with those in the left arm and forearm, could be detected.

There was absolutely no loss of power in the right leg; and the reflexes, both superficial and deep, seemed quite natural and equal in both lower extremities.

There was no perceptible right-sided facial paralysis; but the patient, who had lost all his upper teeth, seemed to have some difficulty (probably not paralytic) in moving the upper lip on both sides. The skin sensibility was markedly impaired in the right forearm and hand, more especially over the fingers and on the outer (extensor) aspect of the forearm. In the upper arm there also appeared to be some loss of tactile sensibility; but there was apparently no anæsthesia in the right leg. The exact testing of the skin sensibility was, however, impossible because of the difficulty which the patient had in expressing himself. The visual acuity seemed equal and natural in the two eyes. Perimeter measurements (made on 11th and 12th March) showed partial right homonymous hemianopsia, with some concentric limitation of the field in the left (seeing) half of each field. (The hemianopsia was probably more marked than is shown in the figure; for the patient, who was very definite and certain in his answers when the left side of the field was being taken, was uncertain and hesitating when the right side of the field was being examined. The parts of the fields coloured black in the charts were absolutely lost.) The pupils were equal and contracted both to light and to accommodation. No abnormal ophthalmoscopic appearances were observed.

Hearing, taste, and smell, so far as they could be tested owing to the aphasic condition of the patient, seemed equal and natural on the two sides. The heart seemed normal; the urine was very pale, its sp. gr. 1010; it was acid, not too copious, and contained on albumen.

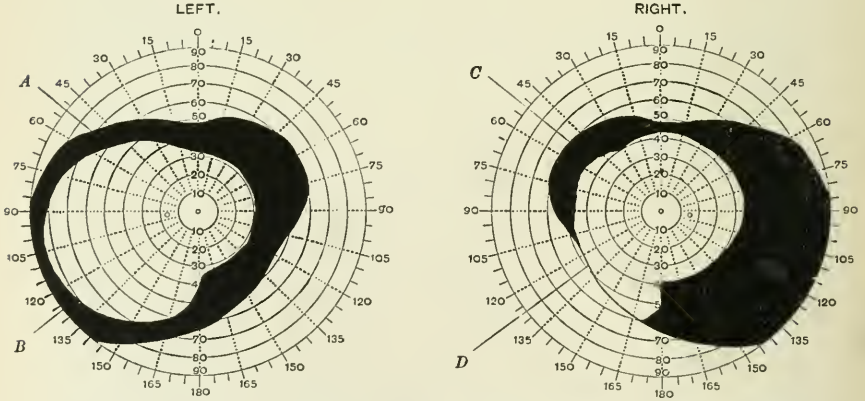


FIG. 1. — *Perimeter Chart of the Fields of Vision for White in the case of J. F., showing Partial Right-sided Homonymous Hemianopsia; Peripheral Constriction of the whole of the left half of the Field in the Left Eye (the parts to which the letters* A *and* B *point), and Contraction of the upper part of the left half of the Field in the Right Eye (the part to which the letter* C *points).*

A high degree of aphasia was present. On analysis it was found to consist of very marked word-blindness, with apparently some degree of motor aphasia (amnesia) in addition.

When the patient first came under observation, I was doubtful if there was not some "word-deafness," or lack of comprehensive (intellectual) power. When asked, for instance, on 14th March whether he was 100 years old, he at once expressed emphatic dissent both by gesture, expression, and verbally, saying, "Oh no." But when asked immediately afterwards if he was 30, he hesitated for some time and finally answered "Yes." Immediately after this, he at once and emphatically said, in answer to questions, he was not 40 and not 50.

This defect, if any, of intelligence in comprehending spoken language, was, however, only temporary; since 21st March I have not observed any mistakes of this kind.

A more careful analysis of the speech condition at the time when the patient first came under observation showed the following facts:—

Motor Functions of Speech: Motor Aphemia.—The patient had lost the power of spontaneously speaking in sentences. He was also unable to name most common objects (such as a knife, pen, key, pencil) which were shown to him, but he always knew what they were, and was at once able to repeat their names when they were mentioned in his hearing. He was able to make a few intelligent replies to questions in single words, and to use the words Yes, No, Oh no, in an intelligent and propositional sense.

Motor Agraphia.—He had lost the power of spontaneously writing in sentences. He had also lost the power of writing to dictation. He was unable to write any letter of the alphabet when told to do so. He could, however, copy both printed and written characters with his left (non-paralyzed) hand quite as well

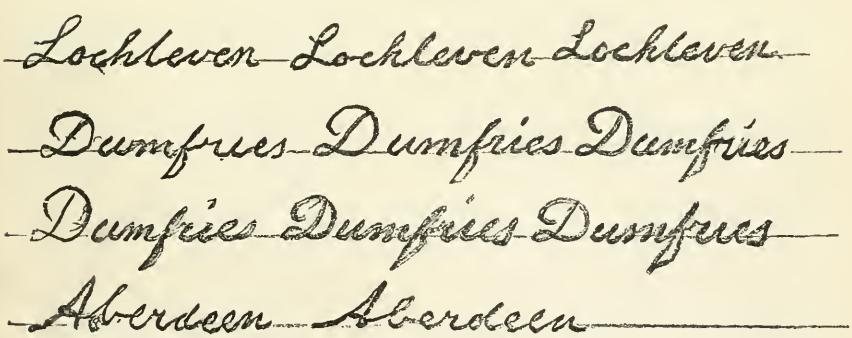


FIG. 2.—*Specimen of Writing copied with the Left Hand by J. F. (the patient whose case is described in the text).*

as most right-handed persons (see Fig. 2). He could also draw with his left hand at least as well as most right-handed persons

(see Figs. 3 and 4). He knew the different parts of the house, which he drew, and although he could not himself name them, he at once pointed correctly to the different parts when they were named to him.

Fig. 3.—*Facsimile of Drawing of a Man's Head, made with the Left Hand by J. F. (the patient whose case is described in the text).*

Sensory Functions of Speech.—With the qualification mentioned above, there seems to be no "word deafness." There is, however, very marked "word blindness." The patient is, as has been already mentioned, well acquainted with ordinary objects (such as a knife, pen, pencil, etc.) which are shown to him, although he cannot name them spontaneously. He also at once recognises pictures and portraits (the Queen, Prince of Wales, Edinburgh Castle, the Scott Monument, etc.) He is, however, unable to recognise a single letter of the alphabet; he can neither spontaneously name the letter when it is shown to him, nor can he, when the names of different letters are repeated aloud to him, indicate when the particular letter, which he is asked to recognise, is named. He is also unable to recognise figures, either spontaneously or when they are named to him.

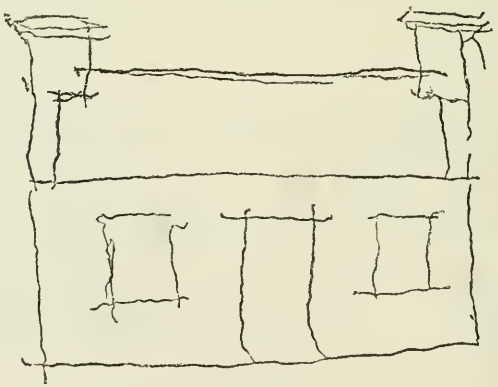


Fig. 4.—*Drawing of a House made with the Left Hand by J. F. (the case of "Word-blindness" described in the text). Drawing reduced from 4 to 2½ inches.*

On the 11th of March, he was asked to name the letters and figures on a calendar for the month which was standing on my table. The result was as seen on the following page.

The letter O is the only one which was correctly named; the T (in THR.) was correctly named, but in SAT. was named N.; the N in both SUN. and MON. were named T.

MARCH—NETOS			
SUN. = CST			
MON. = NOT			
TUE. = QSW			
WED. = TRN	2=R	9=N	16=EU
THR. = TRN			
FRI. = WXR			
SAT. = XRN	5=T	12=NX	19=ON

FIG. 5.—*Copy of a portion of a Calendar for March* 1887, *showing the mistakes made by J. F.*

Numbers were on this occasion named as letters; 2 was called R; 9 N; and so on.

At the same visit he copied the letters TUE correctly, but named them, after he had copied them, STH. He also wrote to dictation, on the same occasion, T for A, S for B, R for C; and when told to write D, E, F, said "I cannot," or "I do not know."

Efforts of this kind, and, indeed, any sustained attempt to speak,

read, etc., were attended with marked flushing of the face, perspiration of the head, and appearance of great effort.

The patient was advised to diligently practise his writing, and to try and read, and learn the alphabet.

Subsequent Progress of the Case.—His general condition and loss of speech slowly and gradually improved. Motor power was to a considerable extent regained in the muscles of the upper arm and forearm; the muscles acting upon the wrist and fingers have, however, remained until the present date, 20th June, absolutely paralyzed. The swelling and moisture of the paralyzed hand have almost altogether disappeared.

On 18th May, spontaneous speaking in sentences was still lost; the patient could now name many more objects than when he first came under observation; and in replying to questions a few more words were made use of.

He was still quite unable to write spontaneously or to dictation. He could not even write his own name, though he made, on this and several other previous occasions, more or less satisfactory attempts to do so (see Fig. 6). He was still, as a rule, unable to write any letter of the alphabet when told to do so.

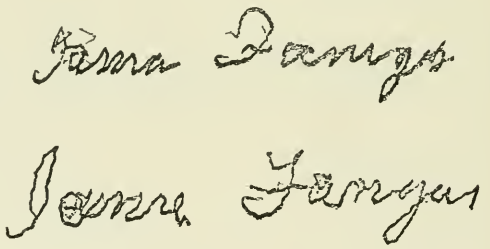


Fig. 6.—*Facsimile representations of Signatures of J. F., made with the Left Hand on two separate occasions.*

At present he cannot write or recognise the letters *o*, *a*, or *s* which I wrote on the black-board, nor does he know these letters when they are named to him. He has recognised these letters on a previous occasion; and I have more than once found that he could write and recognise some letters, which perhaps at the next visit, or if he was tired or agitated, he seemed to be quite unacquainted with. He is now able to write some of the individual letters, such as *a*, *o*, *s*, when told to do so; and if he makes a correct start, and writes the letter *a* to dictation, he is sometimes able to go on and to write many letters correctly and in their right order. On one occasion, he got up to the letter *p* in this way. (See Fig. 7.)

He is, however, quite unable to read any letter after he has written it; he cannot point out the letters *a*, *c*, *o*, for instance, in the line of letters which he has written; and even when the

letter to which his attention is being directed is correctly named to him, he does not recognise that it has been named. In this respect (the power of spontaneous writing some letters, which

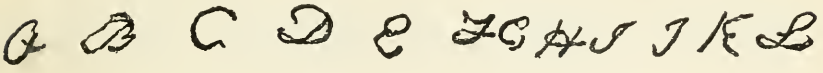


Fig. 7.—*Facsimile of Alphabet written by J. F. on 21st May.*

The alphabet was correctly written up to the letter p. The patient was unable to read any of the letters after he had written them, and did not even recognise them when named to him.

he is quite unable to read after he has written them) the case seems to resemble in an early stage the case of word-blindness described by Westphal, "in which the patient, at first unable to read his own writing, acquired the power of deciphering it by passing the tip of his index-finger over each letter as if he were writing the word a second time."[1]

Soon after the patient first came under my observation, his wife, who always accompanied him when he came to see me, stated that he was able to read and make out some things which interested him in the newspapers. I paid very little attention to this statement; for it is well known that aphasic patients, who are utterly unable to read a single word, not unfrequently occupy themselves with a book, newspaper, or a paper, and appear to their friends to be intelligently interested. Knowing then that this patient was unable to read a single letter of the alphabet, I paid no attention to this statement. When, however, his wife stated on a subsequent occasion, that he had, while walking in the cemetery, read to her the names (with which, be it observed, he had not been previously acquainted) on some of the tombstones quite correctly; and also that he had, on another occasion, turned up and pointed out in a guide-book the name of a landlord about whose property they were talking, my attention was aroused, and I found on investigation that the patient could read many words, the individual letters of which he was totally ignorant of, even when they were named to him. Thus, at present he reads correctly the words which I have written on a piece of paper (see Fig. 8); also the words *house* and *cat*, written in the same way; *book*, he says is *boot*, and *something* he reads as *some one;* the words *hand*, *right*, *left*, he is unable to read.

He is totally ignorant of the individual letters composing the words which he is able to read, and does not know them when they are named. Thus, asked, with regard to the first letter in the word doctor, which he has read correctly—Is it *a*? Is it *b*? Is it *c*? Is it *d*? he fails to give any sign that he connects the sound *d* with the written symbol. And so on with all the other letters.

[1] Quoted by Ross, *Aphasia*, page 75.

His knowledge of figures is now better than it used to be, but still very imperfect.

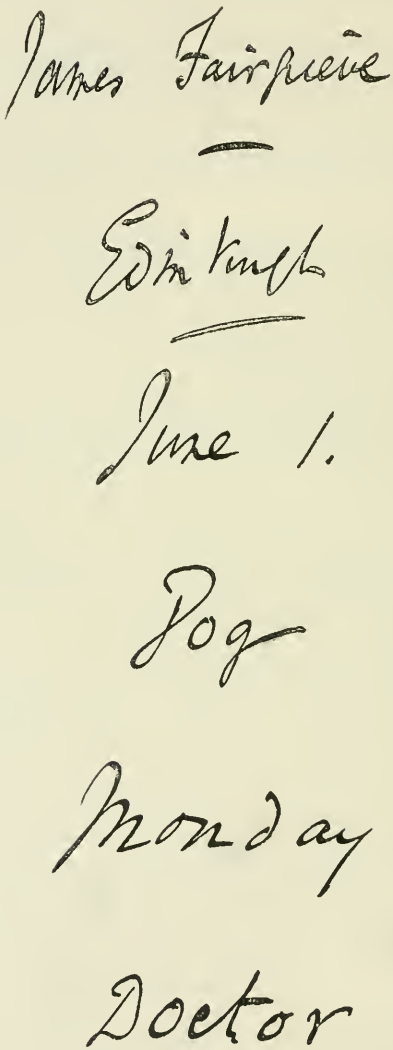


FIG. 8.—*Facsimile Copy of Words read by J. F. on 1st June.*

The patient was unable to recognise any of the individual letters contained in these words, even when they were named to him.

At the end of June, the patient's condition was essentially unchanged; I have had no opportunity of examining him since that time.

W. S., æt. 34, a cooper, first came under my observation in the out-patient department of the Edinburgh Royal Infirmary on 10th February 1886, suffering from the effects of an attack of intra-cranial syphilis, and complaining of pain in the back and loins.

The patient, who looked as if he had known better circumstances, and who gave me the impression of having been both intelligent and fairly well educated, stated that he had contracted syphilis from his wife some six years ago. Four years ago, he had suffered from very severe neuralgic pains in the head, and his right eye had became paralyzed. Three years ago, he had had what he termed an "apoplectic attack," which had been followed by paralysis of the left side of the body—face, arm, and leg. He has not been able to speak well since. He very slowly and gradually recovered from this condition; the paralysis of the face and arm had, he stated, entirely disappeared, but the leg was still weak. He suffered from severe headache before the apoplectic attack came on. For the past four months, he has been able to follow his former employment, and to earn fifteen shillings a week as a cooper. Before his illness he was employed as a foreman cooper, and could read and write well.

The patient's memory was evidently very much impaired, but the essential accuracy of the above statement as to the previous history is confirmed by Dr John Thomson, who had seen the patient a few days after the onset of the hemiplegic attack. Dr Thomson has kindly furnished me with the following note as to the exact mode of commencement of the attack:—"The patient's friends state that, some years ago, he had an attack of inflammation in the side; he has also suffered from rheumatism, but not from rheumatic fever. For the past twelve months he has had neuralgia of the head, which was followed by paralysis. The right eye was shut for sixteen weeks, and he saw double; the rest of his face was not affected, and no other parts of the body were paralyzed. The lid of the right eye has always drooped since the attack. He has also suffered from recurrent attacks of neuralgia in the head. On Sunday morning last (26th January 1883), he choked while sitting at breakfast; he then 'took a shiver,' and was put to bed; there was some difficulty in getting his coat off his left arm. During the afternoon of Sunday, the left side gradually became more paralyzed; he spoke a little when roused, but he has never spoken clearly since. He lay in a stupid condition until Tuesday afternoon, and seemed to have pain in the throat; he was very cross and irritable, and would not swallow. On Tuesday afternoon, he spoke better, but did not move his arm. On Wednesday, he spoke quite intelligently, and was not so irritable; had a dose of castor oil; has refused to take everything since; he attempted to get up and go out; had to be held down in bed, and was therefore brought to the Infirmary."

On examination (*10th Feby.* 1886), the patient was seen to be

well nourished. The gait was markedly paralytic, with much circumduction of the left leg. There was some drooping of the right eye-lid, and the left side of the face seemed somewhat flatter than the right.

On more particular examination, the left leg was seen to be somewhat smaller than the right, but the muscles were nevertheless fairly firm and well nourished. The force which could be exerted by the muscles of the left leg was distinctly less than that which could be exerted by those of the right. The right arm and hand were a little stronger than the left, but not by any means more so than is often seen in right-handed people; and it is important to note, on account of the aphasic symptoms which were present, that this patient had always been right-handed. No perceptible difference could be perceived between the strength of the two buccinator muscles; the conjunctival reflex was very active on the left side; the slight flattening of the left side of the face, with increased depth of the right naso-oral fold, may therefore have been natural to this patient; there was, in short, nothing to show that it was paralytic.

There was some ptosis of the right lid; the patient could neither rotate the eyeball upwards nor downwards; the eyeball was not rotated inwards during efforts of fixation for near objects, but it was moved inwards (*i.e.*, to the left) in association with the opposite eye (*i.e.*, when both eyeballs were turned to the left).

The patient says he sees double when an object is placed above the eye. The right pupil was dilated, and was twice the size of the left; its outline was slightly irregular; it contracted, but not briskly, both to light and accommodation.

Both acuity of vision and the field of vision were considerably impaired in the right eye; much less so in the left. (See Fig. 9.)

On ophthalmoscopic examination, the retina surrounding the margins of the right disc were perhaps a little hazy; the veins in the right eye seemed to be a little larger and more dilated than those in the left, and the disc itself, I thought, was a little paler (possibly, however, this was the result of better illumination, due to the dilated pupil).

The other special senses appeared to be normal.

Condition of the Reflexes.—When the patient first came under observation, a most beautiful knee clonus could be produced with the greatest facility in the left leg; the right knee-jerk was very lively, but no clonus movement could be elicited on this side. A slight ankle clonus was with difficulty produced on the left side.

Tickling the sole produced clonic spasms in the left thigh. The plantar reflex was very lively on the right side.

The cremasteric reflex was exaggerated on both sides, being greater on the *right* than on the left.

The abdominal and epigastric reflexes were very lively on the *right* side; less so on the left.

Lumbar and scapular reflexes were not obtained.

Urination and defæcation were said to be normal.

The sensibility of the skin to tactile and painful impressions seemed normal and equal on both sides of the body, no discoverable difference being observed between the sensibility of skin in the two legs. Sensibility to temperature was not tested.

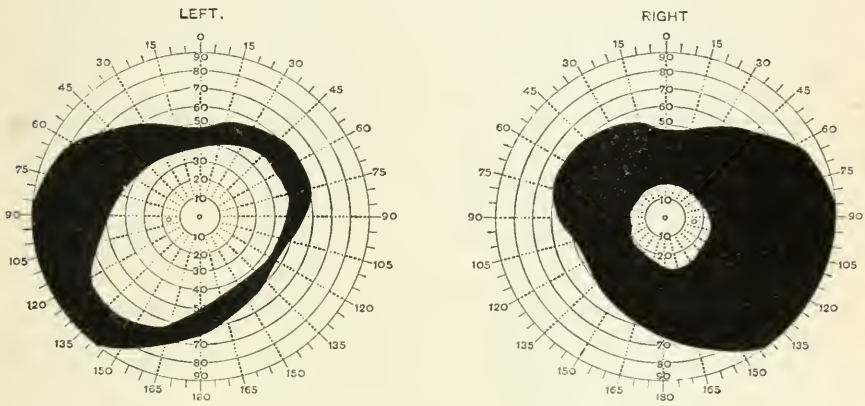


FIG. 9.—*Perimeter Chart of the Fields of Vision for White in the case W. S., showing marked Contraction of the Right, and some Contraction of the Left Field, probably the result of old Papillitis.*

The patient was well nourished. The heart and kidneys appeared to be healthy.

Psychical Functions.—Memory seemed to be much impaired, and the mental functions generally (so far as could be judged from the statements of the patient as to his mental capacity prior to his illness) deteriorated. But although the patient's memory was very defective, and his cerebration generally slow, he could not be termed unintelligent,—he seemed to understand what was said to him, did what he was told, and obeyed all the requests which were made during the course of the examination. His emotional faculties were under loose control; he laughed much too easily, and seemed inordinately amused at many of the tests which were applied in eliciting the foregoing particulars.

During the past fifteen months, the memory and intellect have become still further deteriorated.

Speech Faculties.—There was no motor aphasia; articulation was somewhat slow, thick, and hesitating, being not unlike the blurred articulation produced by drink.

There was no "word deafness," the patient understood everything that was said to him, and obeyed all requests in an intelligent manner.

I find it noted that, when the patient first came under observation, he could read both type and written characters. This statement must, however, be taken with some reserve, for I have no exact notes as to the details of the examination in this respect, and when (after an interval of some months) the patient was again seen and this point was carefully tested, his reading power was much impaired.

On 2nd June 1887, he was able to read type characters from a book fairly well. The words *the, on, of, by, hotel, same, each, Switzerland,* were read correctly. He seemed, however, at the same date almost unable to read written words. The word *the,* written as in Fig. 10, he said was three figures. When asked after a considerable interval to read the same word, he said, "It is two strokes and a nothing." The word *on,* written as in Fig. 10, he read correctly. The word *no* (see Fig. 10) he read as *nothing;* and the word *by* (see Fig. 10) he said, after some hesitation, "It is *b, y;* no, it is *b, e;* no, *by.*"

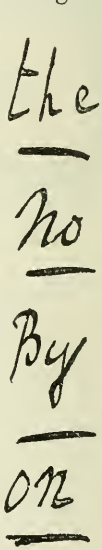


Fig. 10.—*Facsimile of written Words, some of which the patient W. S. was unable to read on 2nd June 1887.*

When tested on another occasion, he was unable to read the written words *the* and *of,* while *by* he said was *baby.* While being tested on this occasion, he was obviously quite conscious of his defects, for he said, "I am like a daft man, before I spell that out now, I forget what I began with." On another occasion, he correctly read "*William Sheels,*" "*Edinburgh,*" and "*How do you feel,*" but could not read the words *dog* and *cat,* when written by me.

He was also unable to write words correctly. His manipulative powers were unimpaired, but he always introduced far too many letters into his words. Even his own name, and the word *Edinburgh,* which must have become by frequent repetition, when he was well, thoroughly "well organized," were hopelessly mis-spelt. A facsimile representation of his signature, and of the word *Edinburgh* as he wrote it, are given in Figure 11.

He made some mistakes in writing individual letters. At one visit, the letters, *a, o, c, g, t, p,* and *e,* were written correctly, but on the same occasion he wrote *w* when told to write *v,* and *v* when told to write *b.* On the same occasion, he wrote *good* for *God; goog* for *dog; tct* for *cat; alon* for *no; htee* for *the;* and *ndam* for *and.*

The most striking defects were, however, in his power of reading and writing numbers.

He was able to read and write single numbers (1 to 9) correctly; beyond this, he was invariably wrong, and the mistakes which he

made were in many cases repeated, although they were not in all cases identically the same.

On different occasions, he wrote the number 10 as follows:—100, 101, 1001, 10001, and 10101.

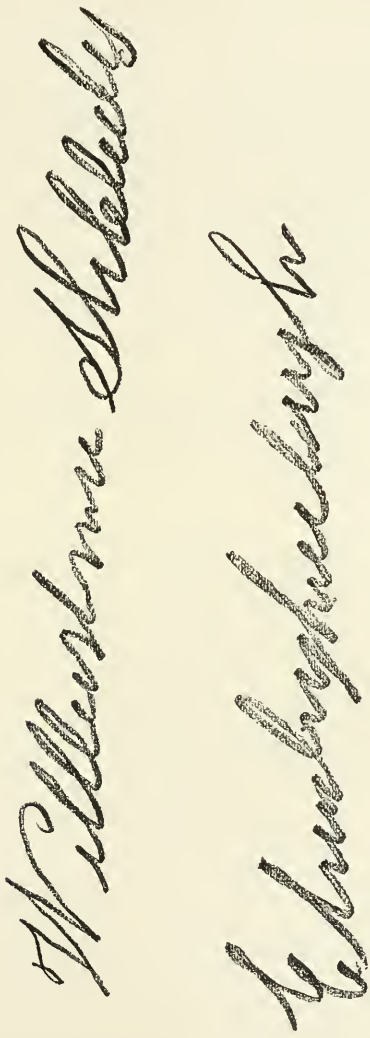


FIG. 11.—*Facsimile of William Sheels' signature, and of the word "Edinburgh," as he wrote it on 2nd June 1887.*

On the 8th of May, 11, 12, 13, 14, 15, 16, and 17, were written 111, 121, 131, 141, 151, 161, and 171.

On the 2nd of June, 14 was written 1141; and 15 was written 501.

The number 100 was written on different occasions as 1001, 10001, 100001.

The number 20 was written on different occasions as 120, 210, 200; the latter mistake was made on two separate days.

On one occasion, 30 was written 300 and 310; 50 was written 1601; 60 was written 115; 70 was written 071; 80 was written 8100.

On the 2nd of June, the number 10 was read as 100; 101 as 10; 1001, which he had previously written as 10 or 100, was not read, but he said, "Oh, it is millions." 201 was read as 300; and before arriving at this result, he said, "That is a two and a one, that is three, that is three hundred." 20 was read as 200; 210 was read as 400; 150 was read as 155; 21 was read as 12; and 13 as 31.

On the 8th of June, 10 was read as 100; 1000 as 110; 101 as 111; and 1001 as 111. On the same occasion, all the single numbers were read correctly. He was then asked to read 15, and said, "Oh, but you are coming it too heavy on me now." When pressed to try and read it he said, "That is one and five, that is six."

In all these mistakes there is, it will be observed, the tendency to put in too many figures, and to end up with the figure 1.

Some of the mistakes, such as 210 being read 400, it seems impossible to account for.

Remarks.—The chief point of interest in this case is undoubtedly the peculiar form of aphasia. It seemed to consist chiefly of an inability to correctly interpret *written* words, and both printed and written numbers. The mistakes in writing words and figures were clearly the result of this derangement in the sensory and intellectual side of the speech mechanism, and were not due to any defect of the way out for written speech ("writing centre").

It is important also to note that this peculiar form of word-blindness was associated with left-sided hemiplegia, and that the patient was right handed. These facts do not, however, conclusively prove that the aphasic condition was the result of a lesion of the right hemisphere. Such a conclusion would be premature, for in cerebral syphilis, as every one knows, it is no uncommon thing to meet with several cerebral lesions in the same patient. Although, then, the grouping of the symptoms is highly suggestive that the aphasia was, in this case, due to a lesion of the *right* hemisphere, yet, in the absence of post-mortem verification, it is impossible to be absolutely certain that it did not depend upon a lesion of the left hemisphere.

The paralysis of the third nerve was clearly due to a local lesion, probably a gumma at the base of the brain, involving the trunk of the third nerve; and there is nothing, so far as I can see, in the clinical history of the case to suggest the presence of a lesion of the left hemisphere. The left hemiplegia was probably,

I think, due to syphilitic disease and subsequent blocking of the middle cerebral artery on the right side. It is possible that the gumma which involved the right third nerve, and produced the paralysis of the ocular muscles, had subsequently extended and involved the right middle cerebral artery. The fact that the paralysis of the face and arm had, for all practical purposes, completely disappeared, while considerable paralysis of the leg remained, showed that the syphilitic vascular lesion had more completely involved (*i.e.*, more completely blocked) the branches of the middle cerebral artery which supply the motor centres for the leg, than those which supply the motor centres for the face and arm.

The form of alternate hemiplegia in which the face, arm, and leg are paralyzed on one (the opposite) side, and the muscles supplied by the third nerve are involved on the other (the same) side, as the lesion, is usually supposed to be indicative of a lesion of the crus cerebri (the hemiplegia being due to arrested function in the fibres of the pyramidal tract supplying the muscles on the opposite side of the body); and in those cases in which the hemiplegia and the paralysis of the muscles of the third nerve are simultaneously and suddenly produced,—in other words, in which there is reason to suppose that the lesion is a hæmorrhage—such is usually, perhaps almost invariably, the case. But when the paralysis is slowly established, and more especially in those cases in which the hemiplegia and the third nerve paralysis are not simultaneously produced, the crus may not be in any way involved.

In the latter group of cases (*i.e.*, where the third nerve paralysis on the same, and the hemiplegia on the opposite side, to the lesion are not simultaneously produced), the two independent paralyses may, of course, be due to two independent lesions.

In other cases of this description, and this is the point I wish to emphasize, for I am not aware that it has previously been noticed, the third nerve paralysis on the same, and the hemiplegia on the opposite side, are due to one and the same lesion at the base of the brain, which is situated in front of *and does not involve the crus cerebri;* the third nerve paralysis being due to a local lesion of the trunk of the nerve, while the hemiplegia is the result of a lesion of the internal carotid or middle cerebral artery. In other words, the hemiplegia is produced by a lesion of the motor centres or of the pyramidal tract in the higher parts of the cerebral hemisphere.

Lesions which produce this form of alternate hemiplegia are, in the great majority of cases, syphilitic. While lesions, which involve the crus, may be acute (hæmorrhagic extravasation into the tissues of the crus, for example) or chronic; in the latter case, the lesion of the crus may, of course, result from a gumma or other lesion at the base of the brain, which presses upon it from without.

III. Exhibition of Specimens.

Mr Miller showed—(*a.*) A RENAL CALCULUS removed by nephrolithotomy. The patient had been under observation in Dr Wylie's ward off and on for five years. His symptoms were very few. He had lumbar pain in the right side, occasional hæmaturia, followed by pain in the bladder from the passage of clots. This continued so steadily that Dr Wylie came to the conclusion that there was a renal calculus. Cutting down on the kidney, and passing his finger along the attached edge, Mr Miller felt a hardness, and passing a needle along his finger struck the calculus, which was got at through an incision through the capsule with a tenotomy knife, and with a little difficulty, from the great mobility of the kidney, the stone was removed by the aid of a lithotomy scoop. It weighed 52 grains. For a few days most of the urine came by the wound. After that it passed naturally, and the wound healed in about ten days. (*b.*) AN EPULIS, which had been growing for seven years, from the alveolar margin of the left upper jaw of a female patient. It was pedunculated, and easily removed.

IV. Exhibition of Instruments.

Dr Foulis showed—(*a.*) AN ASPIRATOR SYRINGE; (*b.*) AN ASEPTIC CATHETER and SMALL FORCE PUMP for washing out the bladder; (*c.*) A MIDWIFERY FORCEPS, in which traction was made by a handle parallel to the axis of the blades, and lying behind the sacrum when the instrument was inserted.

V. Original Communications.

1. ON POISONOUS PRODUCTS IN FOOD-STUFFS.

By R. W. Philip, M.A., M.D., F.R.C.P. Ed.

EVERY one is familiar with the records—and, doubtless, many members of the Society might add to such records from their own experience—of cases where urgent symptoms of poisoning have been unexpectedly induced through the ingestion of ordinary articles of diet. I do not refer to those examples of individual idiosyncrasy in respect of certain foods, which have been long recognised, and of which we have a familiar illustration in the constant supervention, in some persons, of severe urticaria after partaking of such meats as shell-fish, eggs, milk, cheese, various forms of grain and farinaceous foods, and different kinds of fruits and green produce. Among other remarkable illustrations, Lauder Brunton describes (*Disorders of Digestion*, p. 274) the case of a lady in whom a *single* strawberry caused the face to swell up until the eyes became closed. Such idiosyncrasies are exceptional, and,

as a rule, are easily recognised by the subjects of them. In these cases the treatment is obvious.

The remarks which follow have reference rather to a more important group, namely, those cases where the ingestion of some common article of food has been followed, simultaneously in a large number of individuals, by urgent toxic symptoms. Current medical literature contains many such reports, and it is needless to enter into their details. In some instances, the poisoning has followed the use of such simple comestibles as fish, bacon, veal, sausage, etc.

In not a few of these, the suspected articles have been subjected to searching examination, in order to the discovery of mineral or other poison which might be supposed, by accident or otherwise, to have obtained access. But, as a rule, the cause of the toxic phenomena has escaped detection.

The rapid strides which bacteriological research has made within the last few years, and the development side by side with this of the investigation of the group of albumen derivatives which have tentatively been classed together as ptomaines or cadaveric alkaloids, have now given fresh impetus to this line of inquiry, and furnished us with a more tangible guiding-thread in the difficult search. Already some good work has been done; and to a few of the more prominent landmarks in this field I shall have occasion to refer in the remarks that follow the more strictly analytical and experimental part of the present communication.

My attention was first drawn to the subject in connexion with other work on the etiology of the infective processes—an investigation where the combined light obtained from the bacteriological and the more strictly chemical side seems to promise the best results. While that work was in progress, I had, through the kindness of Dr James Ritchie, an opportunity of seeing a remarkable case, where the patient rapidly sank, with many of the usually accepted symptoms of septicæmia, after the ingestion of a large supply of ripe strawberries. I believe the fatal issue was traceable to the grave intestinal disturbance induced in a subject predisposed to gastro-intestinal derangement; for the patient was a moderately strong man, and careful examination into the history of the illness pointed most evidently to the strawberries as the exciting cause of the illness. Unfortunately, we were not able to clear up the matter further, but the probability is great that death was due to some form of ptomaine poisoning. A month or so after that, there occurred the startling case of poisoning on a large scale at Carlisle. Members may recall the accounts of the sudden development of alarmingly toxic symptoms after a wedding breakfast—symptoms which presented themselves more or less in the larger portion of the company—culminating in the death of the bride and the serious illness of a number of the guests. The case has the additional local interest that the chief sufferer died in

Edinburgh. The general character of the symptoms was uniform, and consisted mainly in severe depression and marked gastro-intestinal disturbance.

Perhaps it will give greater vividness to the picture, if I read to the Society the description of the symptoms presented by the two chief figures at the wedding breakfast—the bride and bridegroom—as these are given by Dr Barnes of Carlisle, who has instituted a laborious inquiry into the subject, and whose account is now in course of publication in the *Practitioner*. The bridegroom suffered to a much slighter degree than his wife, who finally succumbed to the attack. Dr Barnes (quoting from Dr Carmichael of Edinburgh) says:—"On Saturday morning, 21st August, I was asked to visit Mr and Mrs L., of Carlisle, who had come to Edinburgh on the previous Thursday afternoon. I found both patients in bed, suffering from vomiting and diarrhœa and griping pains in the belly. The servant in attendance stated that, on their arrival on the previous Thursday, between 3 and 4 P.M., they were both apparently well. The maid did not see them again until Friday morning, when she observed Mrs L. to be sick and ill, complaining of sickness and diarrhœa. . . . . Mr L., on examination, was found to be a well-nourished healthy-looking man, of sound constitution. He looked pale and sick, and complained of griping pains in the belly. He had frequent loose purging of watery, dirty-coloured fluid, vomiting of watery mucus tinged with bile. The skin was cool and perspiring; pulse, 84. Temperature not taken. He was unable to retain any food. On the 22nd, the patient was much in the same condition. On the 23rd, sickness and purging less frequent; griping pains frequently recurring. In the evening the symptoms were greatly relieved." (In addition to these symptoms, Dr Barnes notes that Mr L. suffered from intense thirst, with scanty secretion of urine, and that he also had severe herpes of the lower lip, while, on recovery, general desquamation of the cuticle occurred.)

"Mrs L. appeared very ill when first seen by Dr C. on the morning of the 21st. The surface of the body was cold, the skin perspiring, looked collapsed, eyes sunken, cheeks hollow, complexion ashy and livid-looking, finger nails livid; she had frequent griping pains in the abdomen, vomiting of a dirty, slightly greenish, watery fluid, constant purging of a yellowish watery fluid, which afterwards became of a slightly greenish tinge. Her tongue was red and somewhat dry, with a dirty-looking dorsal fur. The pulse was regular and of fair strength. Temperature, 99°. On examination all the organs appeared sound. On the 22nd, she is reported as having passed a restless night. The vomiting and purging, with pain, continued with little remission; the pulse was 78; temperature, 98°·2. These symptoms continued, with slight variation, until the 24th, when the symptoms of collapse became more marked, and the sphincters gave way. On the 25th, she gradually became worse,

her countenance became dusky, her face slightly flushed; the vomiting ceased, but she had involuntary purging; the motions both yesterday and to-day being very offensive and watery. The temperature was 100°·2, but the pulse began to fail. The conjunctivæ were suffused and the pupils contracted, but shortly before death they became dilated. The heart-sounds remained clear and regular until within three hours of death, which took place between 7 and 8 P.M."

Besides the two, of whose illness the details have been given, fifteen or sixteen persons suffered more or less severely from similar symptoms, directly traceable to something partaken of at the breakfast.

Some difficulty was experienced in fixing on the particular cause from a *menu* that was sufficiently various to allow of the possibility of error. Perhaps it may be well to give the full list.

1. Game pie, containing hare, wood-pigeons, and beefsteak.
2. Boiled salmon, with mayonnaise sauce.
3. Chickens.
4. Boiled ham.
5. Grouse.
6. Lemon whip.
7. Red jelly.
8. Yellow jelly.
9. Trifle, with jelly.
10. Turkish delight.
11. Pine-apple.
12. Melon.
13. Apples.
14. Pears.
15. Walnuts.
16. Grapes.
17. Apricots.
18. Bride's-cake.
19. Cobs or rolls of bread.
20. Tea.
21. Coffee.
22. Champagne.
23. Port.
24. Sherry.

Ultimately the plan was adopted of making a careful inventory of all the dishes consumed, and systematically marking off those which were partaken of by the affected persons and those which were partaken by persons who were present at the *déjeûner* or had tasted the meats, but did not suffer. The inquiries were most carefully conducted, and the individual answers were noted separately, and afterwards compared. In this way the list of possibilities was first of all reduced to six, viz., the bread, the pie, the ham, the jelly, the bride's-cake, and the salmon. Of these the bread and cake were excluded, as they were not partaken of by some who suffered severely, while they were partaken of freely by others who suffered little, if at all. In a similar way the game pie (which had been especially suspected), the salmon, and the jelly were satisfactorily excluded. This concentrated suspicion on the ham, the remains of which were accordingly seized.

Portions of the ham were submitted to various pathologists and analysts for examination. The result of ordinary analysis was negative, and pointed to the absence of any of the usual forms of irritant poisons. Mr Hare was entrusted with a portion for bacteriological examination, and I believe portions were sent to the Pathological Department of the University and to Dr Klein. Mr

Hare was good enough to let me have a portion of what was submitted to him.

It is the results of my examination which I have to communicate, in the hope that, by discussion, fresh light may be thrown on an important subject. Mr Hare will presently give an account of the microscopic results.

Method of Examination.—The portion of ham received weighed 3 gms. It had been soaking for a week or so in 10 cc. absolute alcohol, with a view to microscopic work. The ham was first carefully bruised in the absolute alcohol, and to the mixture there was slowly added three or four times its volume of rectified spirit. The whole was transferred to a flask, covered at the mouth with thin muslin rag. This was exposed for about twenty hours to a gentle moist heat (36°–40° C.) in a Koch's steam sterilizer. The fluid was then carefully filtered three or four times, till the filtrate was perfectly clear. This was measured and evaporated down over a water-bath, at temperature below 50° C., till it was reduced to 1-20th its original bulk. The latter part of the process was conducted slowly, as there was reason to fear that some of the derivatives might be volatile. The resulting syrupy fluid was used for injection.

With a view to controlling the results, a similar weighed piece of healthy ham, purchased from a restaurant opposite to the University, was subjected to a like process, and utilized for contrast injections.

The quantity at our disposal necessitated economy, and I had to content myself with experimenting on frogs, and, to a less degree, on the human subject.

Three sets of experiments were carried out:—

I. Measured quantities of the extract obtained from the suspected ham were injected into the posterior lymph sac of frogs, and similar quantities of the extract from the healthy ham into other frogs. These afforded positive conclusions.

II. Having approximately fixed the smallest quantity of the suspected extract which produced symptoms, a healthy frog received, first of all, this quantity of the extract from the healthy ham, and, after all symptoms must have developed, it received the same amount of the suspected extract.

III. The suspected extract was administered to the human subject by the mouth.

SERIES I.—Injection of measured quantities of the extract from the suspected ham, and similar injections of the extract from the healthy ham into the posterior lymph sac of frogs.

Experiment 1.—℥v. extract from *suspected ham* injected into healthy frog (R. temp.).

12.10 P.M.—*Injection made.*

12.15 P.M.—Frog appears quieter.
12.20 P.M.—Tends to lie prone. Reacts but slightly to mechanical stimulation. No alteration of pupil.
12.25 P.M.—Content to lie on back. No longer attempts to jump when stimulated. Occasionally makes dragging forward movement of hind legs.
12.35 P.M.—In *statu quo.*
12.45 P.M.—Forward crawling movement attempted more frequently. Head still dependent.
12.50 P.M.—Slight improvement. Head more erect.
12.55 P.M.—Return to state of depression.
1.15 A.M.—In *statu quo.*
1.45 P.M.—In *statu quo.*
2.15 P.M.—Remains gravely depressed. No spontaneous movement, but reacts, if irritated, by making attempts at forward leaps.
3.15 P.M.—Slight improvement.
4 P.M.—Activity restored in modified degree. Movements made from time to time are less ready than normally.
6 P.M.—Improvement continues, but movements are still sluggish. Will still lie on back, if placed.
8 P.M.—Gradual improvement.
On following morning seems perfectly restored.

Experiment 2.—℥v. extract from *healthy ham* injected into healthy frog (R. temp.).

12.5 P.M.—*Injection made.*
12.10 P.M.—Jumps about freely.
12.15 P.M.—No change.
12.30 P.M.—In *statu quo.*
12.45 P.M.—In *statu quo.*
1.15 P.M.—Jumps about in lively fashion.

Experiment 3.—℥x. extract from *suspected ham* injected into healthy frog (R. temp.).

11.25 A.M.—*Injection made.*
11.30 A.M.—Has become much quieter. Head tends to droop.
11.35 A.M.—Scarcely reacts to mechanical stimulation. Lies indifferently on back or belly.
11.40 A.M.—In *statu quo.* When irritated, makes slight attempt at crawling movement forwards.
11.45 A.M.—Perfectly still, and almost reactionless.
11.55 A.M.—In *statu quo.*
12.15 P.M.—In *statu quo.*
12.25 P.M.—Occasional attempts at crawling movement forwards.
12.45 P.M.—Depression graver. No effort at movement.
1.15 P.M.—In *statu quo.*

2.15 P.M.—Appears moribund. Respiratory movements scarcely to be detected. Practically reactionless.
3.15 P.M.—In *statu quo.*
4 P.M.—Vitality seems slightly increased. Slightly more evident reaction to mechanical stimulation. Occasionally ineffectual attempts at voluntary motion.
6 P.M.—Still lies indifferently on back or belly, though reacts more readily to strong mechanical stimulation.
8 P.M.—Slight improvement.

On following morning, 10 A.M., found to be still considerably depressed, though it jumps about slowly when stimulated.

Experiment 4.—℥x. extract from *healthy ham* injected into healthy frog (R. temp.).

12.5 P.M.—*Injection made.*
12.10 P.M.—In *statu quo.*
12.15 P.M.—Seems rather quiet. Jumps about less.
12.20 P.M.—Movements more sluggish.
12.25 P.M.—In *statu quo.* When placed on back at once rights itself. Limbs firm, and resist extension. Reflexes appear exaggerated, slight tap on plate giving rise to slight spasmodic movements.
12.30 P.M.—As before.
12.35 P.M.—Slight depression appears to be passing off.
12.45 P.M.—Movements more natural. Tendency to twitching movements absent.
1. P.M.—Appears in normal state.
1.30 P.M.—In *statu quo.*

Experiment 5.—℥xv. extract from *suspected ham* injected into healthy frog (R. temp.).

11.15 A.M.—*Injection made.*
11.20 A.M.—Frog manifestly much depressed.
11.25 A.M.—Lies motionless on belly, with head dependent. No alteration on pupil. Respiratory movements less evident.
11.30 A.M.—In *statu quo.* Occasionally twitching movements of limbs occur.
11.35 A.M.—Depression most grave. Limbs flaccid and placed indifferently in any position.
11.40 A.M.—Appears moribund.
12. A.M.—State of complete torpor. Twitching absent.
12.15 P.M.—No sign of life. Respiratory and head movements absent.

Post-mortem Examination. — The pupils equal; moderately dilated. Limbs flaccid.
Heart in state of diastole.
All chambers engorged. Other organs anæmic.

Experiment 5 A.—℥xv. extract from *suspected ham* injected into healthy frog (R. temp.). This experiment afforded similar results to the last, the frog becoming apparently moribund in 40 minutes after injection; but with this difference, that the state of depression very gradually passed away. For three days the symptoms were traceable.

Experiment 6.—℥xv. extract from *healthy ham* injected into healthy frog (R. temp.).

12.5 P.M.—*Injection made.*
12.10 P.M.—No change.
12.15 P.M.—Frog appears quieter.
12.20 P.M.—More evident signs of depression. Head tends to be dependent, but limbs remain firm and resist extension. If placed on back, soon rights its position. Reflexes normal, or slightly exaggerated.
12.25 P.M.—Depression seems passing off. Movements are slightly crawling in character.
12.35 P.M.—Depression largely gone.
12.50 P.M.—Sits with head erect, in normal position. Occasionally makes voluntary jump.
1.10 P.M.—Jumps about more freely, though legs tend slightly to drag.
1.20 P.M.—Appears to have returned to normal state.
1.50 P.M.—In *statu quo.*

SERIES II.—The smallest quantity of the suspected extract capable of producing symptoms being fixed, a corresponding quantity of the extract from the *healthy ham* was injected into the posterior lymph sac of a frog, and, after all symptoms must have developed, the same amount of the *suspected extract* was injected.

Experiment 7.—Injection of ℥v. extract from *healthy ham* followed, 65 minutes later, by ℥v. extract from suspected ham.

12.5 P.M.—*Injection of healthy extract* (℥v.).
12.10 P.M.—No result.
12.30 P.M.—No result. Jumps about in lively fashion.
12.40 P.M.—In *statu quo.*
12.50 P.M.—In *statu quo.*
1.5 P.M.—Frog as lively as previous to injection.
1.10 P.M.—*Injection of suspected extract* (℥v.).
1.15 P.M.—Frog appears quieter.
1.20 P.M.—Evident depression. Lies flat on belly. Scarcely reacts to mechanical stimulation.
1.25 P.M.—Will lie for time on back when placed. Limbs somewhat flaccid; may be freely extended.
1.30 P.M.—Depression deeper. Reflexes diminished. Limbs,

when extended, only brought back to normal position after an interval of time.

1.40 P.M.—Depression deepening.

2 P.M.—In *statu quo*.

The frog continued in much the same state throughout the afternoon, with gradual improvement towards evening. On the following day it appeared natural, and was returned to the ranarium as well. Two days afterwards it was found dead, with no sign of irritation at seat of injection.

SERIES III.—Administration of the *suspected extract* to the human subject.

Experiment 8.—℥x. of the *suspected extract* were taken in three doses (at intervals of half an hour), diluted with water.[1]

In fifteen minutes after the last dose, headache was experienced. This was followed in half an hour by a distinct feeling of squeamishness, with a slight tendency to faintness, so that the recumbent posture had to be adopted. Shortly afterwards a cramping sensation was felt in the gastro-intestinal tract, with an indication of commencing peristalsis. This continued for an hour and a half, when the patient went to bed, feeling uncomfortable, with a peculiar mawkish taste in the mouth, as of the extract. A restless night was passed, with continuous feeling of nausea. This persisted in the morning, so that the patient remained indoors. During the morning and throughout the day the peristaltic effects were more marked, and induced several calls to stool, when slight diarrhœic motions were passed. As the afternoon wore on the symptoms gradually subsided.

From the above I think it is clear that a poison of some activity was found in the portion of suspected ham examined, and, by presumption, in the ham partaken of. This is supported by the positive result obtained in the three series of experiments with the suspected material, and by the *comparatively* negative results obtained in the control experiments. I say "*comparatively*," because certain symptoms were undoubtedly induced by injection of the healthy extract; but these were, in each instance, trifling in comparison with the others, so that, for the present, they may be left out of account. Possibly they were due to a trace of creasote or other of the agents used in the process of preservation. Excluding that, we must admit the presence of a poison of considerable potency.

Assuming, then, the presence of such a poison, we have to examine as to its probable *origin*.

And here there is abundant scope for theorizing. In the first place, it is not likely that the poison was a mere extraneous addition to the ham, obtained either in the process of cooking or

[1] The report which follows was dictated by the medical man, who subjected himself to experimentation.

of preservation. There was nothing in the appearance or the taste of the ham to awaken suspicion, and an ordinary chemical examination by the local analysts discovered the presence of nothing unusual. I think it more likely that the poison was an *autogenous product.* And this associates it with those other cases of poisoning from the ingestion of various articles; for example, cold veal, corned beef, sausage, which have from time to time been described, and to which reference has been made.

It is conceivable that through some flaw in the preservative methods, or through some accident in cooking, a process of fermentation of a subtle character was permitted, with the toxic product as one of its resultants. And such bodies have now been separated from decomposing fish and other articles used for consumption.

With regard to the present instance, two theories seem to me tenable.

1. Mr Hare will speak presently of the presence of certain micro-organisms in the sections he examined. As to their relation to the tissues, room must be left for individual opinion. But granting that these organisms have a distinct significance, then, I think, we must also admit that the poison, whose properties have been illustrated, was probably the product of the organism.

2. Even though these organisms be regarded as accidental, it is conceivable that, owing to such an accident as I have just suggested, some other microbe or ferment, which our methods have not detected, obtained access and gave origin to the poison either by secretion or by elaboration from the albuminous elements of the tissue.

Such a view has the substantial support of analogy in the physiological process of peptone production, and in various of the fermentations. It has further support from the pathological side, for it will be remembered that, so far back as 1856, Panum demonstrated the production in putrid fluids, from albuminous constituents, of a poison *capable of withstanding boiling.* And the still later researches of Bergmann and Schmiedeberg have shown, that during the decomposition of albuminous bodies, a basic substance is elaborated, possessing definite chemical and physiological properties, capable of inducing symptoms resembling those associated with the process of septicæmia. Further, as is well known, Selmi has established beyond doubt the occurrence of definite alkaloidal bodies during the various stages of putrefaction of albuminous bodies. It is to Selmi that we owe the name ptomaine or cadaveric alkaloid. Selmi's work has been continued by a large school of followers, and now the number of such professedly supposed alkaloids is legion. Of especial importance are the researches of Brieger, who has succeeded in separating from decomposing flesh two distinct alkaloids—neurine and neuridine—and from decomposing fish, muscarine, ethylenediamine,

and gadinine, the first two of which (muscarine and ethylenediamine) are eminently poisonous.

Unfortunately, the infinitesimal quantity at my disposal would not allow further refinement; and I fully admit the difficulty of judging absolutely of results when one has to experiment with an impure substance. From the appearance and behaviour of the product I obtained, I am inclined to think that possibly there was more than one base present, or possibly an acid and a base, as has been found to occur in certain stages in the decomposition of fibrin and fish, and as was long ago shown by Panum in putrid fluids, where he discovered a poison possessed of narcotic properties associated with the septic poison proper.

It appears to me probable, that the poison in the case before us is akin to the already separated septin or sepsin. The depressant symptoms induced in the frogs, and the depressant and choleraic symptoms produced experimentally and accidentally in the human subject, lend support to this view. For it has been shown that if an animal to whom sepsin has been administered lives long enough, or if artificial respiration be kept up, enteritis certainly appears.

There is no need here to speak of the medico-legal importance of such observation. But the thought suggests itself that such a line of investigation may prove of value, from the ætiological point of view, in the explanation of those diarrhœas of obscure origin, generally grouped under the term of cholera morbus.

On the higher value, from the etiological standpoint of such lines of research, I do not at present wish to dwell. I hope, on some early occasion, to give an account of an extended research which is now in progress, and which I think promises to throw some light on the "modus moriendi" in phthisis.

One word should be added as to the practical application of such research in the way of treatment. It is evident, if the conclusion suggested be correct, that successful treatment must not be limited to the mere combating of the diarrhœa and gastro-intestinal disturbances on the usually accepted empirical lines. But recognising that we have to deal with a definite alkaloid of more or less pronounced properties, we must endeavour to combine the more strictly symptomatic treatment with the exhibition of such remedies as may be known to possess antagonistic properties. Thus, I think, it can be shown in relation to one of the infective processes which I have investigated, that the presumptive alkaloidal principle is pharmacologically akin to muscarin. This may or may not be further substantiated. But hypothetically granting the fact, the clear indication is that our best hope in the matter of treatment rests in the administration of some acknowledged antidotal body, such as atropine, which in the instance to which I refer can experimentally be proved to be most efficient.

Appendix, containing report on portion of ham submitted for examination from another case of poisoning which occurred more recently in Glasgow.

Two slices of roll ham, each *circa* $\frac{3}{8}$-inch thick, together weighing 5 oz., were received on 19th January 1887, from Dr J. B. Russell, Medical Officer of Health, Glasgow.

The ham was uncooked, and appeared healthy, with the exception of a small area near the centre of the cut surface of each slice, which was of darker colour. On closer examination, this area was found to be a patch of muscular tissue, surrounded and completely separated from the rest of the lean portion by fat. In consistence, it was markedly softer than the rest of the lean. Its surface area on each side of the two slices measured almost exactly the same,—$1\frac{1}{8}$ inch × $\frac{1}{2}$ inch. It was evidently the cut section of a band of lean meat, which passed, more or less continuously, through the length of the roll. It was, however, in organic connexion with the rest.

The area had a peculiar smell, quite distinct from the normal smell emitted by the other parts. The odour suggested decomposition.

Portions of the apparently healthy lean, and the entire mass of the altered areas, were preserved for more minute investigation.

I. Microscopic examination[1] of portions, similarly hardened in absolute alcohol and stained, when cut, according to Gram's method, revealed most striking differences.

(*a.*) The sections obtained from the apparently unaltered lean showed the characteristic appearances of well-preserved ham meat. At the sides of the section some micrococci were stained of a violet colour, but evidently these were the natural accompaniments of the exposure of the ham previous to examination.

(*b.*) The sections from the apparently altered portions of lean revealed the muscular tissue in a state of advanced disintegration. The sections were cut with much difficulty. In many parts they presented the appearance of a mass of debris. Here and there the striæ were made out with distinctness, but in the major part of the section they were hardly traceable. Micrococci, as in the other case, were present in abundance at the edge of the section; but in addition, in many parts of the field, clumps of micrococci were distinguishable in close relation with the disorganized tissues. They were perfectly distinct in appearance from the rod-like organism associated with ordinary "taint" in ham.

[1] I am indebted to Mr Hare, F.R.C.S.E., for his valuable collaboration in the microscopic examination.

II. Concentrated extracts, prepared according to the method I have followed under similar conditions, were obtained both of the apparently healthy portions and of the suspected portions. These were utilized for the purpose of testing the physiological action of the ingested ham. Various experiments were performed by means of injection of corresponding quantities of each extract into the posterior lymph sac of large, healthy frogs. The results were most distinct. The following condensed record may be taken as sufficiently illustrative.

(*a.*) *Experiments with extract from suspected portion.*

In ten minutes after injection the frog showed unmistakable signs of discomfort; jumped slowly away to corner, where preferred to remain still. This depression gradually got more marked, till the frog ceased to react to ordinary stimulus; lay like a log, indifferently on side or back. The reflexes remained normal, or but slightly depressed throughout. The injection was made at 12 noon. Frog remained in state described throughout afternoon and evening. On following morning (9 A.M.) it was found moribund; cardiac action not detectable. On removing the sternum, the heart was found distended, with faint systolic wave at long interval. The mouth, gullet, and stomach were filled with a considerable amount of tough mucus-like material.

(*b.*) *Experiments with extract from apparently healthy portion.*

The frog was closely observed for some hours after injection. With the exception of a slight amount of depression, which supervened about 15 minutes after inoculation, and passed off completely in other 15 or 20 minutes, no symptoms were induced.

N.B.—The extracts, in each instance, were prepared in exactly the same way, and reduced to the same extract. The amount injected corresponded precisely in both instances.

From the above, it appears to me clear that the band of muscular tissue discovered in the roll of ham was in a state which rendered it quite unfit for human food, and presumably sufficed to produce such symptoms as were observed in the human subject after ingestion.

2. NOTES ON UNUSUAL MICROSCOPIC ELEMENTS IN FOOD-MATERIALS FROM A CASE OF IRRITANT POISONING.

By A. W. Hare, F.R.C.S. Ed., F.R.S.E. (From the Surgical Laboratory, Edinburgh University.)

The materials sent to Professor Chiene's Bacteriological Laboratory for my investigation consisted of two portions of a ham that had been partaken of at a wedding breakfast at Carlisle on the

19th of August 1886. That breakfast was the cause of a widespread attack of irritant poisoning, affecting seventeen persons, one of whom died from its effects. A full account of the symptoms observed, and of the conditions in which the occurrence took place, is found in Dr Henry Barnes's papers on the subject in the *Practitioner* for April, May, and June 1887. In those papers also is to be seen the evidence on which it is clearly shown that the ham in question, and none other of the food materials used at the wedding breakfast, was the exciting cause of the alarming and fatal illness that ensued. In September 1886 I received the first portion of the ham, a block of the muscular substance, free from fat, but traversed by one or two layers of fibrous tissue (intermuscular septa), amounting to about 2 cubic inches of the material. This was already preserved in absolute alcohol at the time of its receipt. Half of the piece of ham and the absolute alcohol in which it was preserved were sent to Dr Philip for his examination, the results of which are stated in the preceding paper.

Preparation of the Material.—The portion of ham retained was at once submitted to microscopical examination. From previous experience in the examination of ham I adopted the following method in preparing it for the microtome and for subsequent staining. It was first immersed in freshly-boiled distilled water for sixteen hours, during which it was from time to time agitated to free it from the alcohol in which it had been preserved. A portion of it was then placed on the stage of the Williams Swift microtome, and frozen—sterilized hydrocele fluid, partially inspissated, being used as an imbedding material. This material has great advantages over the ordinary mucilage mixture in preparing sections for microscopic examination for the presence of microbes, as its transparency is a test of its perfect sterility; whereas the mucilage frequently contains micro-organisms, which render subsequent accuracy of observation impossible. Two series of sections were made, one longitudinal, the other transverse, to the direction of the muscular fibres in the ham. The former only proved satisfactory in the subsequent examination, the transverse sections proving so very friable, except when of considerable thickness, that they afforded no reliable evidence. A second portion of the ham, obtained six weeks later, was treated in the same way; both pieces were further examined alike, and gave identical results.

Staining Method.—In staining the longitudinal sections I found it a great convenience to adopt the method recommended by Dr Byrom Bramwell in the examination of nervous tissues—viz., by placing the section at once on a glass slide as soon as it is cut, and applying the various staining and clearing media in sequence to the specimen thus already *in situ*. In this way the continuity of the tissue in the section is not disturbed, as is so frequently the case

when other methods are adopted. In the first place, some sections were stained with a freshly prepared six per cent. watery solution of methyl violet. On examination with the Zeiss $\frac{1}{12}$ oil immersion lens, with No. III. eyepiece, a number of micro-organisms were at once detected scattered over the specimen. They were seen to be micrococci of large size, but it was difficult to say whether they were situated on the surface or in the substance of the unstained muscular tissue. To obviate this difficulty, and to obtain a clear view of the relation of the organisms to the tissue elements, other sections were stained by Gram's method, using methyl violet as above employed, and eosine as a contrast stain. Eosine, however, is not a good tissue stain; the constituent elements of the section did not become so clearly defined as was desired. Vesuvine was also used, with equally little success. On substituting for these the usual picro-carmine solution, a very satisfactory result was obtained; the muscular fibres, with their transverse striation, now stood out with distinctness, stained of a pale ochre colour—the fibrous strands taking on a deep pink hue. Against this background of a contrasted colour the deeply-stained micrococci stood out in high relief. Their distribution in the tissue of the section was now distinctly to be followed by slightly altering the fine adjustment of the focussing apparatus.

Nature and Distribution of the Micro-Organisms.—The micrococci are present in small numbers; they are of large size, measuring about 2 μ in diameter, and their distribution in the tissue is not uniform. Many of them lie on the free surfaces of the section, for the most part singly, but here and there as diplococci. Others arranged in pairs, singly, or in some specimens in irregular chains, are within the ham substance, lying in the intervals between the muscular fibres. At some points on the edges of the sections a much larger number of the micrococci are congregated, particularly at the points where inter-muscular fibrous septa emerge at the periphery of the ham tissue. From these heapings up of the organisms at the edges, prolongations extend for a variable distance along with the septa into the substance of the section, but the organisms in these tracts become much fewer and less densely packed together as the observation is extended towards the centre of the section. All these micrococci took up the violet stain with avidity, and became deeply tinged with it; from which reaction I believe them to have been in a state of complete vegetative activity at the time of their preservation in absolute alcohol. There was a complete absence of the faintly-stained specimens among them, with which one is familiar in old cultivations of many species of microbes, where a larger or smaller number of defunct organisms are found among those which still show such signs of vital activity, as proliferation, spore formation, or cell division.

Significance of the Microscopical Appearances.—Well-cooked sound ham shows an all but complete freedom from the presence

of microbes. Ham, in which the process of taint has occurred, is found to be pervaded in a greater or less degree by a bacillus morphologically identical with *Bacillus Subtilis*. Micrococci are, therefore, not an usual observation in the microscopic examination of ham. Seeing, then, that in the case under investigation an unusual microscopic appearance of the ham was associated with unusual results of its ingestion, there is an *a priori* probability that these two conditions are in some way related to one another. It was unfortunately impossible to render this probable connexion more than a surmise by proceeding to actual proof. This could have been done had it been possible to isolate the micrococci from the ham tissue, and by separate cultivation and experiment to show that they give rise to an irritating active principle elsewhere, as they may be presumed to have done in the ham. Their preservation in alcohol rendered this impossible, as they were now incapable of further vegetative activity. One can only, therefore, formulate the opinion that these organisms were the cause of the poisoning which occurred, and that not as infective organized particles capable of growth in living tissues, for they are unlike any known form of pathogenic microbe, but as the elaborators of a *ptomaine* capable of producing irritant poisoning. One is driven to this opinion by a very simple process of exclusion, for since the microbic invasion of the ham and its poisonous properties are common features of the case, and neither can be shown to be in essential relation to any other accompanying feature, the natural inference is that their mutual relation is not merely fortuitous. In Dr Philip's paper is a description of the separation of an irritant poison from the ham substance, for which no origin can be suggested, save a fermentative process in the ham itself, singularly bearing out the opinion now arrived at as to the significance of the organisms found in the ham. The mycotic invasion appears to have occurred after the cooking of the ham, for, as already mentioned, the organisms were in a state of active vegetation at the time of their preservation in the alcohol. This would not have been so had they been present prior to the cooking, for they would have been killed in that process. For the same reason is excluded the possibility of their ante-mortem entrance into the tissues, in which case also they would have held some definite *vital relation* to the tissue elements, of which there is no evidence in any of the specimens examined.

The President presumed that they all took an interest in those cases, and had seen the careful and painstaking accounts of them by Dr Barnes in the *Practitioner*. The question was a very interesting one, and had been discussed in a very able manner by these observers. Looking back, he might say there were three stages by which their knowledge regarding such poisons had been advanced. The first was represented by the discovery and selec-

tion by savages of the poison of putrefactive processes for tipping their arrows; the second by the case in which the flesh of a hunted stag was found to be extraordinarily poisonous; and the third was the present stage, in which these poisons were being studied in their germ and alkaloidal aspects. The latter was to him very interesting, and knowing, as he did, something of the work Dr Philip had been doing in connexion with phthisis, he believed the members of the Society would be much impressed by the effect of his researches on that side.

Dr James Ritchie said it was always very unsatisfactory to have to combat an enemy of which they knew neither the nature nor the strength. It would be a great gain if they could know that the symptoms occurring in such cases were due to the production in the system, by ingested organisms, of one of several alkaloids. Having a knowledge of the alkaloid, they might be able to do more than merely to get rid of the offending material, they might administer the proper antidote. The patient referred to by Dr Philip was a strong young man, whose business led him to take hurried meals at irregular times. Several months previously he had dilatation of the stomach and sarcinous vomiting. Under washing out and careful dieting the sarcinæ disappeared, the stomach contracted, and the patient gained in weight and strength. While in the country he partook largely of strawberries, became ill, and returned to town. His stomach was washed out several times, he was carefully dieted, but a few days later he succumbed to some form of septicæmia, after the strawberries, but whether or not in consequence of them Dr Ritchie was not able to say.

Dr Littlejohn said there were many points to which he should like to draw attention, but at that late hour he felt it would be an infliction. At the conclusion of his remarks, Dr Hare referred to the question whether these organisms had entered the tissues ante-mortem, post-mortem, or after the process of cooking. To his mind there must be some difficulty in their getting to the centre of the tissues after cooking. The reference to the influence of sewage had brought to his mind a case that died suddenly in Great King Street in the practice of the late Professor Miller. Several persons suffered, and one died, after partaking of some food. The larder in which the food was kept was a cellar below the level of the street, and it was certain that sewage gas had got into it. He thought a great mistake had been made in allowing the body of the bride from Carlisle to be removed before a post-mortem examination had been made.

Dr Byrom Bramwell observed that it was some weeks after the poisoning occurred that Mr Hare received the portion of ham from Dr Barnes. Could the micrococci have developed between these two occasions? Were the portions of ham put into absolute alcohol at once or not? Portions had been sent to Dr Klein. Could Mr

Hare tell them what Dr Klein's results were, and if they tallied with his own?

Dr Allan Jamieson asked if the medical man who partook of the extract knew of the substance he was dealing with—in short, whether expectant attention had anything to do with the symptoms?

Dr Dods referred to the value of emetics in such cases. In one instance, about twelve hours after the ingestion of some shellfish, poisonous symptoms occurred. Chlorodyne in large doses was tried without any effect on the pain or diarrhœa, but under emetics all symptoms abated.

Mr Cathcart asked if, in view of the facts brought out by Dr Philip, the old and frequent treatment of these cases with castor oil and laudanum might not prove to be thoroughly scientific instead of the merely empirical treatment it was supposed to be?

Dr P. A. Young said it was a popular idea among cooks that meat pies which were allowed to cool produced symptoms of irritant poisoning. This he considered a subject that might be investigated in connexion with the researches of Dr Philip, as popular ideas often contained a grain of truth.

Dr Foulis held that meat pies made without ventilation were apt to produce poisonous symptoms. When he was at school, between fifty and sixty boys were seized with frightful diarrhœa about ten hours after eating copiously of veal pie which had been so made.

Dr Littlejohn held that the want of ventilation had nothing to do with the poisonous effects. It was the cooling of the jelly and the changes that occurred in it.

Dr Philip, in reply to Dr Allan Jamieson, said that had the symptoms which were induced in the human subject lasted only an hour or two, he would have been inclined to accept the theory of expectancy as a possible explanation of their occurrence, but as they continued through the night and well into the following day, he did not think it sufficient. The castor oil and laudanum treatment suggested by Dr Cathcart had a scientific basis, provided that morphia could be shown to be possessed of properties antagonistic to those of the alkaloid in the food.

Dr Hare, in replying, said that the question raised by Dr Bramwell was one of essential importance in determining the origin and significance of the microbe observed in the ham. He understood from Dr Barnes that, after the occurrence of the poisoning, the ham was kept for some days under lock and key, and that when it came to be distinctly suspected, it was cut into strips, which were placed in absolute alcohol, and transmitted to various observers. He had not seen Dr Klein's report upon the portion sent to him for examination, but was informed by Dr Barnes that no pathogenic organisms were found in it. Mr Hare admitted that there was nothing in the microscopic appearances presented by the micrococci that could identify them as specifically deleterious; and further, that in discussing their significance, Professor Greenfield

did not agree with him in thinking they gave evidence of a bacterial invasion of the ham as a whole subsequent to cooking. At the same time, it was of interest to note that in the last remaining fragment of the ham, which he had been fortunate in obtaining latterly, the same microbes were present, and Dr Philip had separated from it a chemical substance with the same action as that found in the first specimen.

Special Meeting.—June 15, 1887.

Dr John Duncan, *Vice-President, in the Chair.*

I. Election of Ordinary Member.

C. C. Teacher, M.B., C.M., was elected an Ordinary Member of the Society.

II. Exhibition of Patients.

1. *Professor Fraser* showed a case of EXOPHTHALMIC GOITRE in which Professor Lister had removed the greater part of thyroid gland ten years ago. The patient was a female, and the operation was performed on the 18th July 1877. Five years before the operation she had been an attendant on a lady who appeared to have been exacting, and whose exactions wore out and often startled the patient. She was, for example, in the habit of ringing with considerable violence a large bell hung in the maid's room, and this particularly during the night. After some time the girl's health broke down. She suffered from palpitations, flushings, and weakness. About eighteen months later her friends noticed that her neck was large, and afterwards that her eyes projected. In little more than two years the complete phenomena of exophthalmic goitre had occurred. The enlargement of the neck increased, and ultimately interfered with the power of swallowing. She had sensations of choking, and at times violent dyspnœa, and on account of these symptoms she came into hospital to be under Professor Lister's treatment. The tumour was removed. In the course of the operation it was found that it was very deep, and that adhesions had formed, rendering its removal by no means easy. Sir Joseph cut away the bulk of the tumour, and afterwards used the sharp spoon to remove adherent portions. The pulse-rate before the operation was about 130. Five days afterwards it had fallen to 72, and the irregularity of the heart's action had disappeared. In a few weeks the exophthalmos had reduced itself very distinctly. The operation, therefore, had relieved all the symptoms of exophthalmic goitre. About eight months afterwards, during a catamenial period, she suffered from general con-

vulsions, which lasted several hours. Then for two or three years she had at each catamenial period an attack of convulsions, which latterly became less severe, until they disappeared. They were not attended by any loss of consciousness. There had, however, been a single attack about two and a half years ago, but none since. These convulsions were interesting in connexion with experimental operations upon dogs and monkeys, in whom myxœdema and convulsive phenomena have been observed after the removal of the thyroid. She showed no evidence of myxœdema, but the thyroid had not been altogether removed, and what appeared to be a part of the isthmus could be discovered on palpation. At the present moment the patient unfortunately suffered from phthisis.

Professor Chiene said he had assisted at the operation, and though an attempt was made to remove the whole gland it could not be done. There was a good deal of hæmorrhage. The lowering of the pulse-rate had impressed itself very strongly on his mind.

The Vice-President thought a small portion remained occupying the isthmus of the gland.

2. *Dr James Carmichael* exhibited a little girl, aged 7, suffering from HEMIPARESIS, who had been under treatment in the Royal Hospital for Sick Children. The leading fact in the case was that the child presented, when she came under observation, well-marked signs of dyskinesis, or hemiataxia, the inco-ordinate movements being limited to the arm and leg, and to certain muscles of these limbs. There was at first no paresis, which had become progressively manifest during the last two weeks. She was a bright, healthy-looking, intelligent child for her years. The mother's information was that about four months before admission she had a severe attack of typhoid fever, from which she apparently made a good recovery. Two months before she came under notice, shaking movements on voluntary effort began to be noticed in the arm and leg; she suffered from headache and recurrent vomiting. Her movements got gradually worse up to the time of admission. On examination, when the child was brought to Hospital, it was found that when lying quiet nothing was apparent, but on attempt at voluntary movement the right arm and leg displayed well-marked ataxic movements. When asked to put out her hand, a series of jerking movements prevented her from grasping the object. It was noted that these movements in the arm chiefly affected the flexor and extensor groups of muscles. The leg was affected by similar motion before it could be advanced to the ground in progression. Common sensation was unimpaired; muscular sense normal; skin organic, and deep reflexes were normal; sight and hearing good; intelligence unimpaired. All the other systems were noted as normal. The patient was kept in bed, and gradually the ataxic movements became less marked. At this time, about two weeks after admis-

sion, there was noted some degree of paresis of affected limbs, which appeared to have been getting progressively worse during the last few days. Since then some slight improvement of ordinary sensation had been noted in the forearm. When touched in the right forearm or fingers it was felt, but referred to the back of the hand. She had vomited occasionally, though less frequently than before. The case presented an interesting and somewhat rare group, or at all events sequence, of symptoms. Cases of hemi-ataxia, associated with or without hemianæsthesia, usually succeeded rather than preceded paresis. Such cases of post-hemiplegic ataxia were recorded by Demeaux, Grasset, and others, and also referred to by Graves and Bastian in their writings. So far as Dr C. had been able to ascertain, he could find no description of a case where the sequence of events as in the present one obtained. Children were liable to hemiplegic seizures from various causes, generally differing from those obtaining in adult life, and it was noted that these were more liable to occur after fevers, and in this case, doubtless, the attack of typhoid from which the child suffered predisposed to the present state. What the exact pathology of the condition might be was uncertain. They knew that in children embolic plugging and minute capillary extravasation were met with. It could not be doubted that some lesion of the motor centre existed, probably in the caudal portion of the external capsule, leading on, possibly, to sclerosis of some portion of the centre.

3. *Dr Byrom Bramwell* showed a case of SENSORY JACKSONIAN EPILEPSY OF THE VISUAL CORTEX.

4. *The Vice-President* showed two cases of TUMOUR OF THE NECK in which he had been obliged, in the course of their removal, to take away large portions of the internal jugular vein. They were both males. A third patient, female, and over 70, in whom the same had been done for sarcoma, had left the Hospital quite well. All had been performed during the last month. The first of the two shown was an old man with a sarcoma reaching from the omohyoid to the angle of the jaw. Three inches of internal jugular and a portion of the hypoglossal nerve were removed. The tongue was drawn to one side, but had partially recovered itself. The pneumogastric and superior laryngeal nerves were exposed and freely handled. He immediately developed a raucous voice and a short cough, which prevented complete healing by the first intention. There was also some retching. The second case was one sent in by Dr Jeffrey of Jedburgh, after consultation with Dr Joseph Bell. Dr Jeffrey had removed an epithelioma from the lower lip about a year and a half ago. It recurred in the glands. A portion of jaw as well as affected soft parts had to be removed. The wound became septic from the entrance of saliva, but healed quickly. He suffered from no after nerve effects,

although gustatory, hypoglossal, and pneumogastric were largely exposed.

5. *Dr Allan Jamieson* showed a case of ECZEMA OF THE PALM treated by a new method. The patient was a young woman. Both palms had been affected, and it did not appear that the affection was due to anything in her occupation. It had been treated in various ways for three years. On the 27th April she went to the Infirmary. Dr Jamieson proceeded to deal with it on the principle of removing entirely the diseased structures and rebuilding the epidermis on a sound foundation. For that purpose salicylic plaster muslin was applied and continued for rather more than three weeks, until the palms had become tender and sore. They presented a pink and tender appearance. They were then treated with equal parts of lead plaster and vaseline, the hands being dipped in hot water each time the ointment was applied. They were now soundly healed. Dr Jamieson considered that this form of eczema was more common in this than in other countries.

III. Exhibition of Specimens.

1. *Dr Littlejohn* showed—(*a.*) A specimen illustrative of WOUNDS OF THE FACE AND HEAD FROM FIREARMS. They all showed a point that had been remarked by Dupuytren, viz., that in cases in which the bullet had passed through the roof of the mouth the lower jaw was usually broken. He could not understand for some time how this could be, but the explanation was simple. When a fracture of the base occurred and the bones were borne outwards, a fracture of the rami would be liable to occur. (*b.*) Three specimens of DUODENAL ULCER. The first was from the body of an old woman who had strangled herself from a low bedpost. At the examination a well-marked ulcer was found just beyond the stomach. There was no perforation. She suffered from symptoms of indigestion usually three hours after taking food. The annoyance caused by this might have impelled her to commit the deed. In the second case a fatal perforation had taken place. The patient was a young man who, shortly after admission to the Calton Jail, complained of pain in the abdomen, for which he was treated by the surgeon, but, in spite of all that could be done for him, became collapsed in a few hours and died. After death it was discovered he was a soldier in the Soudan, and had been in hospital there with what was declared to have been dysentery. He had been given to intemperance. On opening the body an ulcer of the duodenum was found. The third specimen was an ulcer about the size of a florin from the body of a man who had been dismissed two days before from the Royal Infirmary after undergoing treatment for an attack of delirium tremens. He had

gone out apparently quite well, but shortly after had been attacked by urgent vomiting, which continued till he died. Post-mortem examination showed the ulcer, but did not reveal the presence of any of the more common poisons in his stomach.

2. *Dr Cotterill* showed for *Prof. Annandale*—(*a.*) A TUMOUR OF THE BLADDER from a man aged 68. He had been sent to hospital supposed to be suffering from stone, the chief symptom being hæmorrhage, which was constant and profuse. Mr Annandale removed a small portion of the growth with a lithotrite. The man continued to bleed freely and died of collapse. There was found a blood clot lying on the surface of the tumour at the autopsy. (*b.*) A fimbriated condition of the SYNOVIAL MEMBRANE OF THE KNEE-JOINT of a young man for which excision was performed. (*c.*) Part of a FEMORAL ARTERY which had ulcerated, causing a severe hæmorrhage. The ulceration was due to a large abscess of the thigh, secondary to an axe wound of the knee-joint, which had suppurated. Amputation of the thigh was performed.

3. *Prof. Chiene* showed recent additions to the Surgical Museum of the University of Edinburgh—(*a.*) DUODENAL EXTRAVASATIONS from a woman aged 79, who died of pneumonia five days after receiving extensive burns, chiefly of the third degree, of the arms, shoulders, and chest. (*b.*) Three preparations illustrating certain points with regard to the pathology of varicose veins. The first was a TRANSVERSE SECTION OF THE LEG, presented by Prof. Cunningham of Dublin. It demonstrated that when the superficial veins were enlarged the deep were also affected. The second was a mass of VARICOSE VEINS injected after excision. The patient, a man aged 29, had his leg run over by a cart wheel five years before, and a large bunch of varicose veins formed at the seat of injury. The third consisted of INJECTED HÆMORRHOIDS, obtained post-mortem from the body of a man who died of surgical kidney. This specimen after injection showed the correctness of Whitehead's observation, that internal hæmorrhoids were distinctly localized, for within an inch of the anus the mucous membrane was perfectly healthy. (*c.*) Three NEUROMATA. The first was from the stump of a man, aged 30. He had had amputation performed when 4 years of age by Prof. Spence, and since that time had been operated on on six occasions for painful stump. Portions of nerves were removed on the last of these. The symptoms having recurred, Prof. Chiene removed what remained of the femur, a good deal of muscle, and a neuroma of the sciatic. The other two were neuromata of the ulnar and sciatic nerves respectively, the latter having been sent by Dr Maclaren of Carlisle. It was removed from a boy aged 10, and weighed 1 lb. 14 oz. In the centre of both was a softened cavity, sections from which showed very cellular fibrous tissue. (*d.*) Two specimens of CANCELLATED

EXOSTOSIS, one from the tibia growing from the junction of the internal and posterior surfaces three inches below the epiphysis, the other from the posterior extremity of the spine of the scapula. (*e.*) Two TUMOURS OF THE LOWER JAW, one of which was cystic. In both cases the bone was divided before the mouth was cut into, preventing blood from entering it till the end of the operation. (*f.*) AN EPITHELIOMA OF THE TONGUE removed after a preliminary tracheotomy from a man who was also the subject of syphilis. After the tracheotomy the tracheal tube was connected by rubber tubing with a funnel, the mouth of which was covered with a piece of flannel on which the chloroform was dropped. (*g.*) Two TUMOURS OF THE MAMMA, one a rapidly growing cysto-fibroma, the other a schirrhus cancer of the breast, from an old lady of 82, showing calcification at one part. (*h.*) A LARYNX CONTAINING A FOREIGN BODY, from Dr Adams of Pathhead. He was called to see a child aged 6 months, who was said to be suffering from croup. He found it dead, and on making an examination found a piece of eggshell in the larynx. (*i.*) A COLUMNAR EPITHELIOMA OF THE RECTUM. (*j.*) A PORTION OF BOWEL, showing a CONSTRICTION. Within the last two years he had had two cases in which after operation for strangulated hernia on women there had existed pain and difficulty in defæcation. Both had died some months after the operation, and in one a post-mortem examination had been obtained. At the point where evidently the hernial protrusion had occurred, there was a constriction of the bowel. (*k.*) A SARCOMA OF THE CLAVICLE. The bone was divided before being excised, making an easier operation. (*l.*) Two specimens of SARCOMA OF THE SYNOVIAL MEMBRANE OF THE KNEE-JOINT. One of these was from Prof. Spence's museum, the other had been removed by Prof. Chiene. Both were for some time considered to be cases of chronic rheumatic arthritis, and treated accordingly till their true nature was discovered (in Mr Chiene's case by a secondary growth appearing in the groin), when amputation was performed. (*m.*) The BACILLUS SCARLATINA and CULTIVATIONS.

The Vice-President said the specimen of constriction of the bowel was very like one in which he had to perform enterectomy for obstruction lasting about a week. The constricted portion had become gangrenous, and was removed. The patient died of shock, being exhausted at the time.

Dr Allan Jamieson said he had attended an old gentleman, aged 75, who was always very anxious about the state of his bowels, being distressed if he did not have a daily evacuation. He twice suffered from obstruction, which was overcome, but a recurrence proved fatal. An examination showed that just above the ileo-cæcal valve the lumen of the bowel was no greater than the little finger. This appeared to be congenital, and might in some way account for the old gentleman's anxiety about his bowel.

4. *Dr D. M. Moir* showed for Dr Maclaren — (*a.*) Two CALVARIA from cases of very severe fracture of the skull. (*b.*) A SPECIMEN from a case of cut throat, where although neither the larynx nor pharynx were injured, yet the infiltration of blood and serum had penetrated beneath the lining membrane of larynx and trachea, so as to occasion a fatal œdema of glottidis on the evening of the second day. These circumstances suggested the propriety of performing tracheotomy as a prophylactic measure at the time the vessels were tied and the wound adjusted.

5. *Dr Byrom Bramwell* showed—(*a.*) A large RENAL CALCULUS removed post-mortem; (*b.*) A PERFORATING ULCER OF THE FOOT, with microscopical sections and drawings showing peripheral neuritis; (*c.*) A PORTION OF INTESTINE from a case of ACUTE STRANGULATION, in which the symptoms closely resembled those of irritant poisoning.

6. *Mr F. M. Caird* showed, for Dr M'Bride, a NASO-PHARYNGEAL TUMOUR removed by the aid of the rhinoscopic mirror and Jarvis's snare. The patient was accustomed for a few days to the introduction of the rhinoscopic mirror. Cocaine was painted over the pharynx and posterior nares before the operation. The patient held the tongue depressor himself, while the operator, introducing the mirror with his left hand and the snare with his right, caught the tumour in a loop of piano wire, and removed it with much greater ease than had been done on previous occasions when the snare was introduced by the nose.

7. *Dr James Carmichael* showed an OINTMENT composed of salicylic acid one part, and lanoline three parts, with three drachms of oil of eucalyptus added. He had found it very useful in removing the hard skin on the palms and soles which so often prolonged the convalescence from scarlet fever. The ointment should not be used to the other parts of the body. He had found that it loosened the hard cuticle of the palms and soles very rapidly.

Dr Allan Jamieson said that he had employed for the same purpose an ointment of resorcin, of the strength of one drachm of resorcin, lanoline six drachms, and olive or sesame oil two drachms.

IV. On the motion of Professor Chiene, a Committee of Investigation, consisting of Prof. Grainger Stewart (convener), Prof. Greenfield, Drs J. Carmichael, Woodhead, and Philip, and Mr Hare, was appointed to examine the work which had been done by Drs Allan Jamieson and Edington, to make control experiments, and to report to the meeting to be held on the 20th July.

Meeting IX.—July 6, 1887.

Professor GRAINGER STEWART, *President, in the Chair.*

I. EXHIBITION OF PATIENTS.

1. *Professor Grainger Stewart* introduced a patient who had suffered from BRONCHIECTASIS, and on whom Mr Caird had operated with marked advantage. K. M., male, æt. 27, was admitted to the Royal Infirmary on 14th March 1887, suffering from cough and profuse expectoration of the most offensive character. About nine years before he had a severe attack of pleurisy. Before that time he had been healthy, but after this illness his general health suffered, cough became more frequent, his sputum more copious and fœtid, and his mental powers, probably never bright, declined, until he became little better than an idiot. On admission he was emaciated and generally in bad health, his breath was offensive, and he coughed frequently and expectorated an enormous amount of the most abominably fœtid sputum, the odour of which could only be partially disguised by the continual evaporation of carbolic acid and eucalyptus oil in the room where he slept. Large doses of terebene were also given internally, but with no effect in diminishing the fœtor or on the state of the lungs. The left side of the chest was much contracted, less rounded, and with less movement than the right side; the percussion note was dull anteriorly, and at the apex and base posteriorly; over the middle region of the lung posteriorly the note was more or less tympanitic, and most markedly so just above and internal to the lower angle of the scapula. At this point, too, the auscultatory signs of a cavity were very marked, the breathing was loudly amphoric with marked pectoriloquy, and there were moist sounds on coughing. These latter were, however, most marked toward the base of the lung, and there, as well as at the apex, the breathing was bronchial. Anteriorly, in the third and fourth interspaces, to the left of the cardiac area and at the same level in the axilla, the breathing was cavernous and the vocal resonance increased. There was no alteration in the physical signs up to the date of the operation. The view taken of the case was that the morbid action had originated in pleurisy, and probably chronic pneumonia; that this had resulted in cirrhosis of the lung with dense adhesions, and subsequent bronchiectasis. In these circumstances, and as medicine proved unavailing, Professor Stewart came to the conclusion that surgical interference was warranted. He had the benefit of Mr Caird's advice and co-operation, and it was resolved to try the effect of incision. He left to Mr Caird to describe the process followed, but had much pleasure in stating that from

the time of operation the fœtor had disappeared, the recovery had been fair, and the discharge by the tube was gradually diminishing. The mental condition had also distinctly improved. He thought that the channel should be kept open, so as to drain the cavity yet for a considerable time.

Mr Caird stated that the operative interference was carried out on the 13th April, following the lines indicated by Mr Godlee in his lectures. Chloroform having been administered, the needle of an aspirator was entered below the angle of the scapula, but without result. It was again introduced outside the angle, when a cavity containing most fœtid pus was reached. A free incision enabled one to force a pair of sinus forceps alongside of the needle, which was then withdrawn, and the track thus formed was dilated gradually until, by the resection of about two inches of rib, the finger could be entered. A dependent counter-opening, in order to admit of free drainage, was also made; large-sized rubber tubes were inserted, and the cavity was washed out with warm boracic acid lotion. About a fortnight after the operation, three smart hæmorrhages took place from the interior of the cavity, which gave rise to some little anxiety. Otherwise the progress was satisfactory in the extreme.

2. *Dr Byrom Bramwell* showed a boy, aged 17, who was a striking example of the condition termed ATHETOSIS. There was well-marked right-sided hemiplegia, which involved the arm more than the leg, and muscles acting upon the hand and fingers more than those acting upon the elbow and shoulder. The absence of rigidity and the other characters of the paralysis were, in Dr Bramwell's opinion, suggestive of a lesion either at the time of birth or during early life. The patient stated that the paralysis had existed as long as he could remember. He had no friends, and he knew nothing of his birth. The athetosis movements were very typical, the muscles of the forearm were not so well developed as those on the same side, but were much more developed than one would have expected, considering the amount of the paralysis, the relatively increased size being apparently due to the incessant muscular action; or, in other words, due to the same cause which produces muscular hypertrophy in some cases of this description. Similar (athetosis) movements were present, though to a much less degree, in the toes. There was no hemianæsthesia, no evidence of cardiac or other disease likely to have produced hemiplegia.

II. EXHIBITION OF SPECIMENS.

1. *Dr R. A. Lundie* exhibited a specimen of SCIRRHUS OF LEFT KIDNEY. The patient from whom the tumour was removed was a gentleman, aged 41, who had suffered for about four years from occasional hæmaturia, sometimes copious. The first attack was

followed by severe pain, resembling that attending the passage of a renal calculus, but no calculus was found. No pain was experienced subsequently. About two years after the first hæmaturia, the patient first noticed that his general health was falling off, and that he was losing flesh; he was able, however, to continue his ordinary occupation till about six months before his death. The immediate cause of death was an attack of acute nephritis in the right kidney. A week before death there were the ordinary physical signs of tumour of the left kidney, somewhat obscured by ascites; the descending colon was found on percussion at the outer side of the tumour, and the spleen above it in the infra-axillary region. The ascitic fluid contained only leucocytes and granular debris, thus giving no help in the diagnosis. The tumour had previously been supposed to be enlarged spleen. Post-mortem the tumour was found to be covered by distended veins, as the abdomen had been during life. There were slight old adhesions between the tumour and the spleen, and between a few of the coils of intestine. Except along the course of the splenic vessels, where there was considerable tumour growth, the new formation was entirely limited by the renal capsule. The kidney was removed with the spleen and the portion of descending colon in relation to it. The whole mass weighed 7 lbs. 10 oz. On section, no kidney tissue could be seen, but the infundibulæ were conspicuous, enormously enlarged, and filled with decolorized blood-clot. Under the microscope the tumour presented all the characters of scirrhus. The right kidney was slightly enlarged, and in a state of intense inflammation. There was no sign or symptom during life of disease in other organs. Only the abdomen was examined post-mortem.

2. *Dr James Ritchie* showed several specimens of ARSENICAL WALL PAPERS. He stated that recently, within a period of eighteen months, he had happened to have met with three cases of poisoning by means of such papers. As the opinion seemed to be prevalent that arsenic is not now used in the manufacture of paperhangings, he had ventured to show the Society specimens of these, with the results of their examination by means of Reinsch's process. The symptoms were in all cases those of gastro-intestinal irritation, persistent or frequently recurring. No. 1 included two specimens which were sent at his request by a lady who, with her mother, had some months previously changed their dwelling. They both suffered from similar symptoms, viz., debility, sickness, headache, and diarrhœa. The mother, who rarely left the house, suffered most; the daughter when she went from home recovered, but on returning relapsed. Dr Ritchie understood that the drains had recently been put into good order; from the symptoms he suspected the wall papers. One of them contained a large quantity of arsenic, the other considerably less. The second specimens

involved the health of three not very robust children. After a change of abode they enjoyed better health than formerly, but after about a year they began to suffer from sickness, furred tongue, and occasionally feverish attacks. These illnesses were of frequent occurrence, and sometimes of long duration. Both nurseries (day and night) were hung with a glazed paper, of which the ground was yellowish-gray, with a small green figure sparsely scattered over it. The glaze was worn off the lower parts of the paper. On examination it was found that the gray ground as well as the green figure contained a large quantity of arsenic. The previous occupants of the house were not similarly affected, having apparently been protected by the glaze on the paper, which was unbroken during their occupancy. The third case involved the health of two children, who suffered in the same way as the last, but for a shorter period, as the cause of their illness was discovered earlier. With the exception of one child, all the invalids recovered rapidly after removal from the source of irritation. Several other specimens were also shown. These were obtained at a paper-hanging warehouse. Two of them were yellowish-gray, one blue, and the others contained green. Two of these, at least, were warranted free from arsenic. Dr Ritchie stated that during the same period he had three cases illustrative of another of the less common causes of gastro-intestinal disturbance, which, with the Society's permission, he would shortly relate. He had no pathological specimens to show; they were very unsavoury, and the cause of death in each specimen was probably poison. The cases referred to had great similarity in type, viz., more or less of sore throat, foul tongue, sickness, diarrhœa, and considerable constitutional depression. All the patients were ladies, who were previously not in robust health. The first lady, when recovering from bronchial catarrh with asthma, began without known cause to have sore throat, sickness, diarrhœa, irregular and rapid action of the heart; the asthma returned, and there was marked nervous prostration. The second patient, suffering from chronic rheumatism, was, in consequence of a subacute exacerbation, recommended to rest for a few days in bed; instead of improving, she began to suffer from sore throat, sickness, diarrhœa, and constitutional debility. The third was confined to her room by a feverish cold. Symptoms similar to the last were developed, but the condition of the throat was more variable and more severe. All occurred during the cool weather; in each case an unpleasant odour gradually manifested itself in the sick-room, which was traced to the presence behind the woodwork of a dead mouse. In none of the cases was there any improvement till the patient was removed to another room, and in all the recovery was slow. In each case the mouse was afterwards discovered, two being very large, and all of them moist and powerfully odoriferous. One of the ladies knew that her neighbours had put down poison for mice. In case 2 the drains were

not in perfect order, but in the others no cause could be discovered except the one referred to. It was a question whether the patients would have suffered in the same degree had they been in robust health. It was very noteworthy that the symptoms commenced some days before any odour was perceived.

3. *Dr Byrom Bramwell* showed—(*a.*) CARDIOGRAMS and SPHYGMOGRAMS from a case of aortic regurgitation, in which a marked presystolic thrill and (slighter but distinct) presystolic murmur were audible in the mitral area, but in which Dr Bramwell believed there was no mitral stenosis. This opinion was chiefly based upon the character of the tracings. Dr Bramwell, in describing the case, showed a number of other cardiographic and sphygmographic tracings from cases of aortic regurgitation and mitral disease. He also entered into a detailed description of the views of the late Dr Austin Flint and others on this question. (*b.*) A series of PHOTOGRAPHS, MICROSCOPICAL SECTIONS, and DRAWINGS from cases of CEREBRAL SYPHILIS, illustrative of gummatous syphilis, syphilitic meningitis, syphilitic neuritis, and syphilitic disease of the cerebral vessels.

4. *Mr Hare* showed a series of casts illustrating SURGICAL TOPOGRAPHY OF CRANIUM.

III. Original Communications.

1. THE ADENOID TISSUE AT THE BASE OF THE TONGUE AS A FACTOR OF THROAT SYMPTOMS.

By P. M'BRIDE, M.D., F.R.C.P. Ed., F.R.S.E., Surgeon to the Ear and Throat Department Royal Infirmary, and Lecturer on Diseases of the Ear and Throat, Edinburgh School of Medicine.

THE subject to which I propose to direct attention is, if not entirely new, at least of sufficient novelty to deserve consideration, more especially as to the best of my knowledge it has not been touched upon by any British writer. It has long been known that the base of the tongue, or, in other words, that part of its surface which is in intimate relation with the epiglottis, contains adenoid tissue more or less analogous in structure to that which is found in the faucial and pharyngeal tonsils. It is further of interest to note, that just as in the centre of the pharyngeal vault is situated an orifice leading to a cavity (the Bursa pharyngea), so in the tongue between the circumvallate papillæ and the epiglottis we find the foramen cæcum. As Waldeyer[1] and others have pointed out, there is a complete ring of adenoid tissue, beginning at the vault of the pharynx, passing over the region of the Eustachian tubes to the

[1] *Deutsche Med. Wochenschrift*, 20, 1884.

faucial tonsils, or tonsils proper, and finally extending from here on to the dorsum of the tongue. It is a curious fact that hypertrophy of this lingual tonsil has been apparently so much overlooked as a possible cause of throat symptoms. That this oversight has been more apparent than real will, I think, be seen further on. The only systematic paper on the subject is one which appeared towards the close of last year, and which was written by Swain with the assistance of Hagen, whose clinical journals were placed at the author's disposal.[1] Without wishing to underrate the value of this contribution, it is only fair to others to state that hypertrophy of the lingual tonsil had been observed long before the appearance of the work in question. Of previous observers Swain only refers to Curtis,[2] who described a case of loss of ability to use the voice for any length of time, owing to severe pain in the throat, being caused by attempts at continuous phonation. This symptom was due to hypertrophy of the glandular tissue at the base of the tongue, and was cured by operative treatment. European observers had, however, described symptoms due to a similar cause so far back as 1877. In the *Berliner Klinische Wochenschrift*[3] of that year, Heymann states that Stoerk has described a case in which interference with respiration was caused by the epiglottis being impeded in its movements and irritated by the hypertrophied glandular tissue on the base of the tongue, and Heymann adds that he himself has observed a similar case.[4] The last-named authority has been kind enough to write me that since the period referred to he has observed about a dozen cases of interference with the mobility of the epiglottis arising from the same cause.

In 1879 Betz published a brief but important record of a case,[5] in which glandular hypertrophy of the tongue produced the feeling of a foreign body in the throat. In this case the condition was so marked that the epiglottis actually became caught in the glandular tissue, and the discomfort was completely relieved by cauterization of the hypertrophied mass.

The only text-book in which, so far as I am aware, a reference to this interesting subject is to be found, is Stoerk's *Klinik der Krankheiten des Kehlkopfes*,[6] where he states that in women the margin of the epiglottis is sometimes caught in the hypertrophied adenoid tissue at the base of the tongue, and that fits of coughing and laryngeal spasm may be so produced.

Dr Michael of Hamburg has with rare generosity furnished me with details of two cases observed by him, which, although un-

[1] *Deutsches Archiv für Klin. Med.*, 1886, p. 504.
[2] *New York Medical Record*, 8th Nov. 1884.
[3] No. 52, p. 764.
[4] It is stated that neither Heymann nor Stoerk could find the reference to the case referred to.
[5] *Monatsschrift für Ohrenheilkunde*, 2, 1879.
[6] P. 227.

published, he has given me permission to reproduce, and in doing so I shall translate the words of his letter:—

"1. Frau B. has complained for some weeks of the feeling of a foreign body in the throat. Laryngoscopic examination shows that the epiglottis is caught in the base of the tongue. The epiglottis was raised with a probe, and the discomfort disappeared. The symptoms have not reappeared." (Observed February 1883; letter dated 8th April 1886.)

"2. Fraulein S. complains of difficulty in singing, and a feeling of a foreign body in the throat. The laryngoscope shows that the epiglottis is caught in the mucosa at the base of the tongue. Lifting up the epiglottis has only a momentary effect, as the lingual tissues are much hypertrophied. After repeated applications of the galvanic cautery the hypertrophy of the tongue disappeared, and the epiglottis was permanently freed." (Observed July 1885.)

Only one other observation remains to be noticed. As an "Unusual Cause of Cough," Rice[1] describes fixation of the epiglottis in the hypertrophied papillæ at the base of the tongue; and he is in accord with other observers when he recommends local treatment directed towards the lingual tonsil.

As before said, however, Swain's paper is by far the most elaborate, and, indeed, the only systematic work on this subject. It is based on 190 cases observed by Professor Hagen, who first had his attention directed to the matter six years ago. Some valuable general conclusions could thus be drawn from an analysis of these records. Thus it seems that, while the condition more often produces symptoms in women, yet it exists almost as frequently in men; further, it appears that the affection is one more likely to occur after adult life than before it. In the main, the symptoms observed by Swain and Hagen were those which have been already indicated, namely, feeling of a foreign body, pain usually localized, but sometimes radiating to other parts, and fatigue in speaking. This last symptom Hagen explains by the fact that the base of the tongue and epiglottis undergo constant movement during phonation; but if this motion be impeded by friction between the epiglottis and hypertrophied lingual tonsil, then discomfort and fatigue must result. To these symptoms we have only to add the occasional occurrence of cough and spasm, as described by Rice and Stoerk, and we have before us a fairly complete list of the symptoms that may be met with.

We must now, however, turn to the laryngoscopic diagnosis of the condition in question. Normally, as every one is aware, we see on examination a free interval between the epiglottis and the base of the tongue. If, however, the latter be studded over with hypertrophied tissue, we see this free interval, as it were, filled up. To my mind, the great difficulty is to say exactly

[1] *New York Medical Record*, 1st May 1886.

what is a normal condition of the dorsum of the tongue. I think the following may commonly be taken as an indication of hypertrophy of the lymphoid tissue, viz., contact between the tip of the epiglottis and the base of the tongue, without undue antero-posterior folding of the epiglottis on itself. It is noteworthy that while in extreme cases contact between the margin of the epiglottis and lymphoid tissue is marked even when the tongue is extended, in cases where the hypertrophy is but slight the objective appearances can be best appreciated when the mirror is inserted while the tongue is retained between the teeth. The appearance of the hypertrophied lingual tonsil varies. In marked cases I have generally noticed that the two sides were unequally affected, and the parts had an œdematous look, not unlike the appearance presented by the hypertrophied pharyngeal tonsil.

So far as my own experience goes, the symptoms most commonly produced are pricking and a feeling of a foreign body in the throat —these subjective phenomena being increased if the patient draws back the tongue as in gaping. The explanation of these symptoms is simple enough. Under normal circumstances the margin of the epiglottis is free. When the lymphoid tissue of the tongue is so enlarged as to come in contact with, or even to overlap it, then— two parts being in contact which do not usually touch each other —the subjective sensation of a foreign body results, accompanied or not by pain according to the amount of irritation produced. According to Swain,[1] patients occasionally complain of pain shooting up to the ears, or refer their discomfort to the stomach, larynx, trachea, and intra-scapular region. This is, perhaps, the best opportunity for referring to the most recent article on the subject under discussion. It comes from the pen of Seifert,[2] who, besides confirming the views of Swain, calls particular attention to recent observations which go to show how difficult it is for a patient to localize the pathological causes of clinical phenomena when situated in the throat.

Before referring shortly to treatment and describing one of the most typical cases I have observed, I would utter a word of warning. I do not think that hypertrophy of the glandular tissue at the base of the tongue, sufficient to cause distressing symptoms, is very common, and I am far from believing, as suggested by Swain, that this is frequently the actual cause of the subjective phenomenon known as globus hystericus. On the contrary, I have seen a case in which the condition, *i.e.*, hypertrophy of the lingual tonsil, existed to a slight degree, and in which the patient was an anæmic woman. Local treatment, however, had no effect at all, whereas the old-fashioned but effective administration of Blaud's pills very soon produced a cure. I merely mention this case to show that while hypertrophy of

[1] *Op. cit.*

[2] *Berlin. Klin. Wochens.*, 9th May 1887.

the lingual tonsil is a condition requiring local treatment, yet its recognition has by no means eliminated the necessity for considering certain throat symptoms as purely neurotic. Had Swain been acquainted with the early observations on the subject and with the fact that the affection had been noticed by isolated observers during almost a decade, he would have hesitated before he suggested that hysterical throat symptoms are as rare as he ventures to hint. With regard to treatment, Swain has found good results from the application of a solution of iodine and iodide of potassium in glycerine. The galvanic cautery, too, may be used to destroy the redundant tissue. For my own part, I have so far employed nitrate of silver or chromic acid fused on a roughened probe and applied under guidance of the laryngeal mirror.

I shall terminate this paper by a brief record of a typical case.

A strong healthy young man consulted me in the autumn of 1886. Seven weeks before his visit he had a cold with pain on swallowing. Since that time he had constantly suffered from pricking in his throat and a feeling as if a foreign body had lodged in the right side of it. Examination with the laryngoscope showed the larynx to be normal, but the epiglottis, especially on the right side, was caught in the hypertrophied œdematous glandular tissue of the dorsum linguæ. A probe was introduced between tongue and epiglottis, and a slight amount of force was necessary to free the latter. This produced immediate relief, which lasted for about half an hour.

At his second visit the laryngoscope showed the right margin of the epiglottis to be free, but the patient again suffered from uncomfortable sensations, which returned soon after his last visit (half an hour, as above). On using the laryngoscope, without protruding the tongue, the swollen portion on the right side was again seen to be in contact with the epiglottis. The hypertrophied tissue was then cauterized with solid nitrate of silver fused on to a bent probe. This produced some little aggravation of the symptoms for a short time, and then complete cessation of discomfort for two or three days, after which interval I saw him again. The same treatment was repeated five or six times at intervals of a few days, with the result that the discomfort entirely disappeared as the hypertrophy diminished. The only further point of interest in connexion with the case was, that even at his last visit, after all his symptoms had disappeared, the patient could, by gaping or drawing back his tongue, reproduce them.

It will be seen, then, that in well-marked cases the diagnosis of the affection is not difficult, but it is otherwise when the pathological change is not marked and when the symptoms are vague. In such cases it is well, if contact between the dorsum linguæ and epiglottis be suspected as the cause of irritation, to paint the upper surface of the epiglottis and the corresponding part of the

tongue with a strong solution of cocain. If our suspicions be correct, this proceeding will often temporarily relieve the symptoms. A few days ago I was in this way enabled to obtain indications for treatment, which resulted in relieving a patient who had suffered from distressing throat symptoms for some years.

Dr M'Kenzie Johnston thought the paper brought before the Society was one of great interest, and that in his communication Dr M'Bride had described a real and definite condition, the recognition and treatment of which was of importance to the comfort of the patient. He looked upon the disease as probably similar to the hypertrophic form of follicular pharyngitis. He believed that in the affection known as granular disease of the eyelid, they had an analogous condition, and possibly from the comparison they might get some suggestions as to the pathology and treatment of the disease under consideration. Oculists told them that the condition in the eye favoured the appearance of acute conjunctivitis, and so probably lymphoid tissue might predispose the larynx to acute inflammation. He had seen this association of the two conditions in one or two cases. There were one or two points further that he wished to draw attention to. He was inclined to think that Dr M'Bride had not allowed sufficient importance to general treatment, while rightly laying stress upon local treatment. From the similar conditions found in the eye and pharynx, and which it was admitted were associated with anæmia or lowered vitality, he thought that a course of iron or other tonics was of quite as much importance as the local treatment. He had a case under his care just now where there was marked anæmia, and in which he was trying iron. Dr M'Bride said the disease was one of rarity, and so probably it was in its severer form; but he ventured to think that a milder condition was commoner than was supposed. Many of those cases where patients complained of foreign bodies—fish-bones, hairs, etc., in the throat, or other anomalous sensations, and which were often relieved by local and tonic treatment—would, he fancied, on investigation prove to be more or less marked forms of this follicular inflammation at the base of the tongue. From his limited experience of such cases, he wished to make these suggestions, and not to speak too confidently on a subject which had so recently begun to attract attention, thanks to Dr M'Bride.

Dr M'Bride would like it to be understood that he believed the symptoms he had described in connexion with lymphoid hypertrophy of the tongue might arise in two classes of cases, to wit,—(1.) Robust individuals in whom the hypertrophy is marked. (2.) Individuals with undue irritability of the nervous system (whether due to anæmia or other cause), and very slight hypertrophy, which but for the undue sensibility of the parts would not be appreciated by the patient. Dr M'Bride thought that he had

made this clear in his paper, and even quoted a case of the second variety, which, while resisting local treatment, readily yielded to the administration of iron.

2. *Dr J. M. Cotterill* read a note on the CONDITION OF STIFF GREAT TOE IN ADOLESCENTS, of which the following is a short abstract: — The condition, as discussed in several papers by various surgeons, was known by the following symptoms:—(*a.*) Apparent flexion of the first phalanx of the toe on its metatarsal bone. (*b.*) Inability to dorsiflex the toe, more or less complete. (*c.*) Pain felt in efforts to dorsiflex the toe, as, for instance, in the act of standing on tiptoe. This pain is usually felt over dorsal aspect of joint; at a later stage it may be most marked below the joint, or it may be absent throughout the whole course of the case. Mr Cotterill criticised the views expressed by many surgeons as to the causation of this disease. Amongst others the following causes have been given:—(1.) Injury followed by contraction (Davies-Colley). (2.) Improperly fitting boots (several surgeons.) (3.) Gout, rheumatism, or rheumatoid arthritis (various). (4.) The development of puberty (Howard Marsh). (5.) A defect in the development of the spinal cord (Nunn). (6.) Contractions of various muscles (Ellis). (7.) Contractions of certain ligaments (Anderson.) (8.) Flat-foot (Golding Bird). Mr Cotterill proceeded to demonstrate that, while some of the above causes might lead to stiffness in the joint and inability to dorsiflex the toe, that none of them satisfactorily accounted for the condition as above defined, in which the hallux lies at an altered angle to its metatarsal bone (1st symptom). His view of the case is that the condition which he has named "Hallux Rigidus" is brought about by a necessary combination of flat-foot and boot pressure. The various stages of hallux rigidus and the appropriate treatment for each were then discussed. The following is an abstract of a few of the chief propositions made by Mr Cotterill:—1. Hallux rigidus proper is due to the invariable combination of flat-foot and boot pressure. 2. That while no amount of flat-foot alone will cause hallux rigidus (*c. f.*, as in the case of negroes, who, being frequently flat-footed, never suffer from it), a slight amount of flat-foot in combination with boot confinement is sufficient to bring it about. 3. That hallux rigidus, partly consisting as it does in a more or less complete limitation of dorsiflexion of the hallux to a right angle with its metatarsal bone (which is the normal range of dorsiflexion in the healthy adolescent), exists, *to a certain extent*, in almost *all* cases of flat-foot occurring in persons wearing ordinary European boots. 4. That flat-foot occurring in barefooted nations, or in those who wear sabots or other such boots as provide for ample dorsiflexion of the great toe, does not cause hallux rigidus. 5. That the connexion between flat-foot and hallux rigidus has been frequently denied or overlooked owing to the imperfect

means of testing flat-foot,—and that while an inspection of the foot, or of the footprint, are apt to mislead, the most reliable test is an inspection of the boots which have been worn. 6. That hallux rigidus is frequently one of the earliest manifestations of flat-foot in booted nations. 7. That while it is commonest in young boys, it occurs frequently in girls. (This has been denied.) 8. That in the early stages hallux rigidus may be cured by attention to the flat-foot, and by giving room for dorsiflexion of the hallux in the boot. In the later stages excision of the proximal end of the first phalanx is probably the only means of cure, though palliative treatment by fixation of the joint may give relief to pain in walking.

Surgeon-Major Black remarked that the subject of deformities of the foot was of much interest to army surgeons engaged in the recruiting service, and that the diagrams of the different shapes of plantar sole on the floor would be of value to that department. Mr Cotterill's observations on the form and relations of the ball of the great toe seemed to support the practice of ladies in using a high-heeled boot, and to be somewhat at variance with professional objections to it, as impairing the maintenance of the arch and proper planting of the foot.

Dr Scott Lang mentioned that this subject was brought forward three months ago by Mr Lucy and Mr Davies-Colley simultaneously, but independently of each other. Mr Lucy wrote that he would be thankful to receive any suggestions as to treatment, etc.; and he (Dr Scott Lang) had written to him direct, pointing out that he had overlooked the tendency to flat-foot which was almost invariably present, and that he should support the in-step. He had no wish to differ from Mr Cotterill, and was quite alive to the potency of the other factor (viz., the boot). Still, it seemed rather unsafe to assert that the condition was absolutely impossible in barefooted races, for a case might yet turn up. But, letting that pass, he still contended that of the two factors insisted on by Mr Cotterill, the tendency to flat-foot, or a modification of flat-foot, ought to be looked upon as the primary factor, and the other as accidental or secondary. Mr Cotterill had stated that the boot-maker was the savage here, but the bootmaker might reply that he made the boots of proper length until the tendency to flat-foot, with lengthening of inner edge of the foot, began to come on. The two factors mentioned could at any rate be *considered* separately, and he contended that, even upon Mr Cotterill's own showing, the lengthening of the inner edge of the foot was the primary factor. Confusion arose owing to there being no exact definition of what was meant by flat-foot; but he thought the complaint under consideration was in the main a modification of flat-foot, consisting of undue pressure upwards on the ball of the great toe. The meta-

tarsal bone was extended on the tarsus, and the phalanges and metatarsal bone came to occupy the same straight line, preventing the extensor proprius hallucis from acting. Mr Cotterill ascribed his success in the treatment of his cases to be mainly due to the recognition of a tendency to flat-foot, or a modification of it.

Mr Cathcart pointed out that Surgeon-Major Black was mistaken in supposing that high heels prevented flat-foot, and showed that they actually tended to produce that deformity, by throwing a greater strain on the arch of the foot. He thought that Mr Cotterill had been too exclusive in associating the rigidity of the great toe invariably with flat-foot, and thought that it might be accounted for by the irritation caused by the wrinkle of the upper leather coming on to the great toe joint, perhaps combined with a too short boot. He felt that Mr Cotterill had demonstrated the necessary association of flat-foot with the wearing of boots, but thought that a similar result might be brought about by ill-fitting boots without the necessary accompaniment of flat-foot.

Mr Macdonald Brown, in thanking Mr Cotterill for his able paper, said that the Society owed him a debt of gratitude for the lucid manner in which he had placed before it the various morbid conditions affecting the metatarso-phalangeal joint of the great toe. Up till the present time these had been confused and mistaken by different authors, and in his opinion Mr Cotterill had made out a good case for his "Hallux Rigidus." There was an anatomical point in connexion with the joint in question which, he was glad to notice, had been hinted at in the paper, viz., the condition of the so-called "glenoid," or plantar ligament. This, as was well known, was fibro-cartilaginous in nature, and any part which it played in the contracture (one of the most prominent features of the disease) must therefore be an extremely slight one. Mr Brown would venture to suggest an additional factor in the production of this contracture: the hallucine metatarso-phalangeal differed from the corresponding joints of the other toes not only in its greater size, but also in its cavity, consisting of a horizontal as well as a vertical part, the former being considerably the larger, and having in addition to ligaments, etc., sesamoid bones in its floor. In "hallux rigidus" this secondary synovial pouch must be crushed out of place as well as inflamed, and its cavity if not obliterated at least much altered. This, together with the subsequent contraction of the inflamed subserous tissue, would be capable of aiding in the production of the deformity.

Dr Peel Ritchie, while agreeing generally with the explanation given by Mr Cotterill of the combined effects of flat-sole and pressure, also thought that the form as well as the length of the boot contributed to produce the result. The pointed form of boot was objectionable, as it caused pressure on the joint.

Dr Cotterill, in reply, reminded some of the speakers that he

had specially guarded himself by saying that injury, gout, contractions of muscles, and other such causes, might undoubtedly cause stiffness in the joint of the great toe, but these causes could not possibly bring about the alteration of that angle of 30° of dorsiflexion which normally exists between the hallux and its metatarsal bone, and which is interfered with in all true cases of hallux rigidus as he had defined it to them, and as it had been described in the various papers on the subject. In reply to Mr Scott Lang, Mr Cotterill reminded the meeting that he had brought forward conclusive evidence to show that no amount of flat-foot, however extreme, could possibly cause hallux rigidus in the absence of boot confinement, and that therefore it could not be truly said that the condition was due to flat-foot.

Special Meeting.—July 20, 1887.

Professor GRAINGER STEWART, *President, in the Chair.*

Interim Report of Committee appointed by the Medico-Chirurgical Society of Edinburgh, in terms of the Minutes of the Meeting of the 15th June 1887.

YOUR committee reports that, in accordance with instructions received, an inquiry and experimental investigation has been commenced with regard to the results obtained by Mr Edington, M.B., and published in the joint paper by Dr Allan Jamieson and himself, entitled "An Investigation into the Nature of the Contagium of Scarlet Fever" (*Brit. Med. Jour.*, 11th June 1887).

It will be evident to the Society that sufficient time has not yet elapsed for your committee to have formed a definite opinion upon the intricate and important issues raised; many of the experiments are still in progress, while the number completed is too limited to warrant distinct conclusions.

Before detailing the lines adopted in the inquiry, the committee has to record its appreciation of the courteous co-operation of Mr Edington in supplying information and affording facilities for the furtherance of the work.

So far as your committee has been able to judge from inquiry and observation, Mr Edington appears to have followed carefully the recognised methods of bacteriological investigation, and to have observed the precautions necessary to ensure accuracy in his laboratory work. Evidence has, moreover, been afforded of the large amount of laborious and long-continued work which his research has involved.

While it is impossible to report definite results, it appears

desirable that the Society should be made acquainted with the general line adopted in the inquiry. This has included the consideration of the following points among others:—

1. A preliminary inquiry, with the object of defining accurately the conditions observed by Mr Edington in his research.

2. An inquiry into the characteristics and specific value of the several organisms which Mr Edington has separated and described, with inoculation experiments on healthy animals.

3. The repetition of Mr Edington's observations, including the microscopic examination of scarlatinal blood and desquamation, cultivation experiments with the same materials, and a collateral series of observations on the healthy subject, to control the results obtained.

In the further prosecution of the inquiry, the committee will be much helped by the co-operation of the members of the Society in giving them access to early cases of disease suitable for experimental investigation, especially to cases in the earliest days of the fever, or to those which may be supposed to be in the stage of incubation.

On behalf of the committee,

T. Grainger Stewart, *Convener.*

Dr Allan Jamieson said—In opening the debate this evening on Scarlet Fever, while I am conscious that there are many here present by whom the task might have been more adequately performed, still, since owing to circumstances I have been placed rather closely in contact with a large number of the sufferers in the recent epidemic, certain features of the disease have been brought prominently under my notice; and while many points here undeveloped will occur to others, there are some to which it appears desirable to refer. My remarks are in the main based on the statistics of 200 of the cases admitted into the City Hospital since my appointment. In the selection of these the only item in common to all was, that the list comprises solely such as were taken in not later than the second day of the disease, so far as a pretty careful inquiry could determine. The act of removal, however well accomplished, always causes, at least during the first few days of illness, a rise of temperature and a variable amount of general systemic disturbance. These, however, are of less consequence on the first or second day than on the third or fourth.

Viewing scarlet fever as a whole, it offers in many particulars marked differences from the other exanthemata. Whether the contagious material has been inhaled, swallowed, or has gained access by means of a wound, there is, with one as yet doubtful exception, no variation in the sequence of symptoms. The exception I allude to is that nephritis seems proportionally more frequent in those who have acquired the disease through the medium of milk. A possible explanation of this will be offered

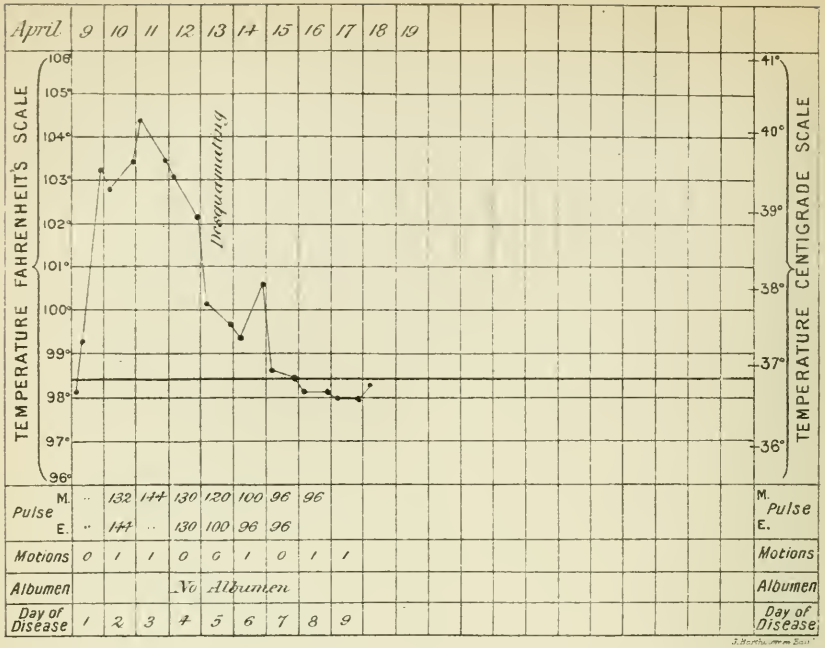
by-and-by. The incubation period is of the briefest. While this is usually stated as from one to six days, there is reason to believe that the later of these dates—those, namely, in which four, five, or six days have elapsed since exposure—are really instances of delayed infection. The poison has been received into the body, but is still outside the circulation. Once in the blood, evidences of constitutional disturbance of no ordinary type are not long in declaring themselves. There are vomiting or rigors, sore throat, then an exanthem; and one who was apparently well on the morning of to-day, may be covered with the rash and in imminent danger of his life within the next thirty-six hours, or may even have succumbed to the virulence of the malady before the eruption had time to become visible. The contagium of scarlet fever expends its primary force rapidly. Thus in eleven cases the highest temperature was reached on the first day, in seventy-six on the second, in seventy-five on the third, in thirty-six on the fourth, and in only two on the fifth. When the date of the highest temperature is later than the fifth day, or when the temperature has not fallen considerably by that day, some early complication, or some constitutional peculiarity, keeps it up. These Charts illustrate this first point. In Chart No. 1 we observe the rapid rise on the first day, the acme reached on the morning of the third, and the normal level regained by the seventh. Chart No. 2 exhibits a similar rise and a more gradual fall. In Chart No. 3 the highest temperature is attained on the evening of the second day, the natural recovered on the seventh. The second rise is due in this case to constipation, which again occasioned the third and smaller elevation.[1]

At a varying period after the subsidence of the rash, desquamation commences, the earliest evidence being usually shown on the tender skin at the sides of the neck, or more rarely between the fingers. It may begin as soon as the fourth day, or the sixteenth even may be reached ere it can be discovered. Such extremes are, however, uncommon. Thus in 1 instance it began on the fourth day, in 1 also on the fifth, in 10 on the sixth, in 28 on the seventh, in 40 on the eighth, in 27 on the ninth, in 41 on the tenth, in 26 on the eleventh, in 10 on the twelfth, in 7 on the thirteenth, in 6 on the fourteenth, in 1 on the fifteenth, and in 2 on the sixteenth. Nearly all the cases in which it was observed first on or after the twelfth day presented some pyrexial complication, and of these rheumatism was the most frequent, occurring in seven of sixteen examples; enlarged cervical glands and secondary sore throat seemed the cause of delay in two others. Desquamation is invariable, and

[1] It is well worth consideration how accurately the temperature in its rise and fall in an uncomplicated example of scarlet fever corresponds with the life-history of the bacillus scarlatinæ as described by Dr Edington, and in particular with its disappearance from the blood after the third day of the disease.

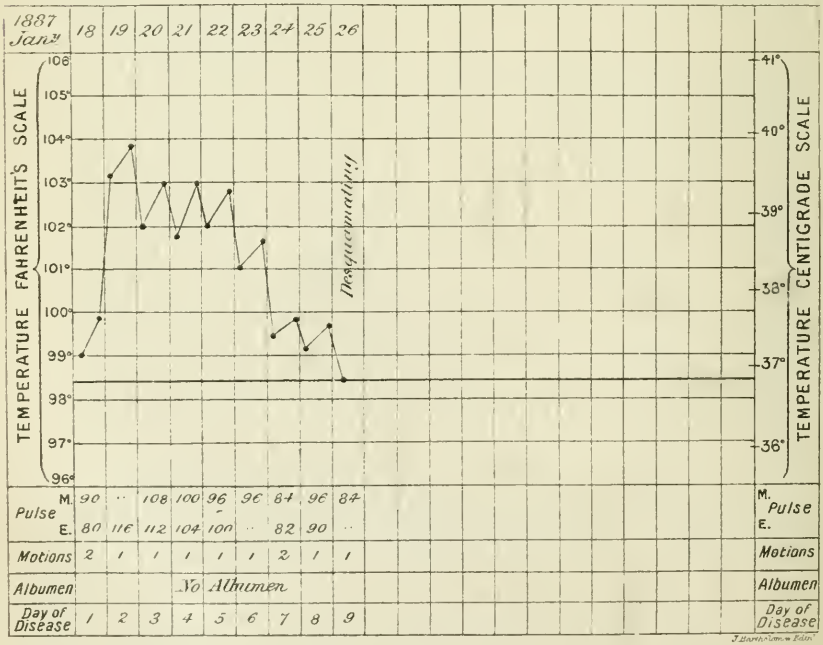
1.

Name *A. J* Age *20.* Disease *Scarlatina.* Result *Recovered.*



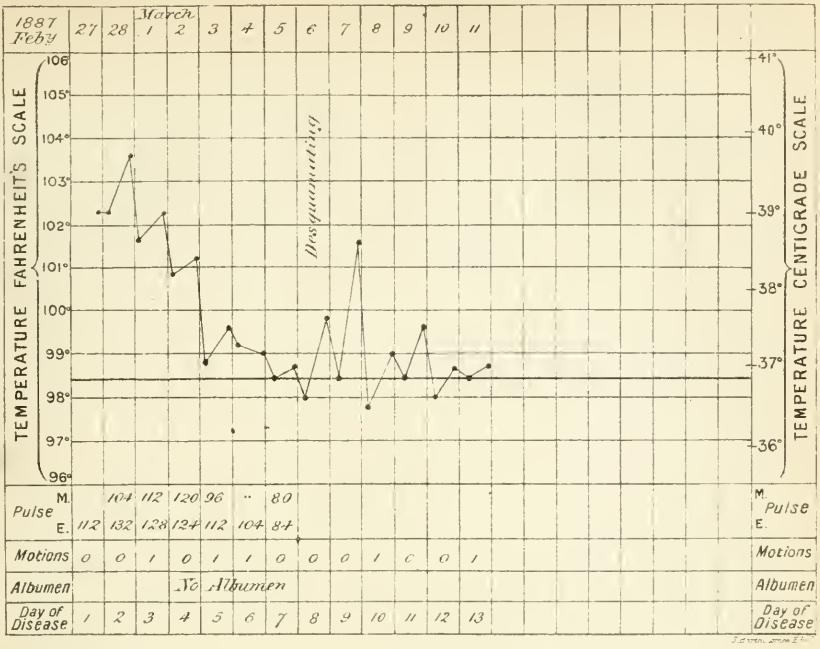
2.

Name *N. G.* Age *23.* Disease *Scarlatina.* Result *Recovered.*



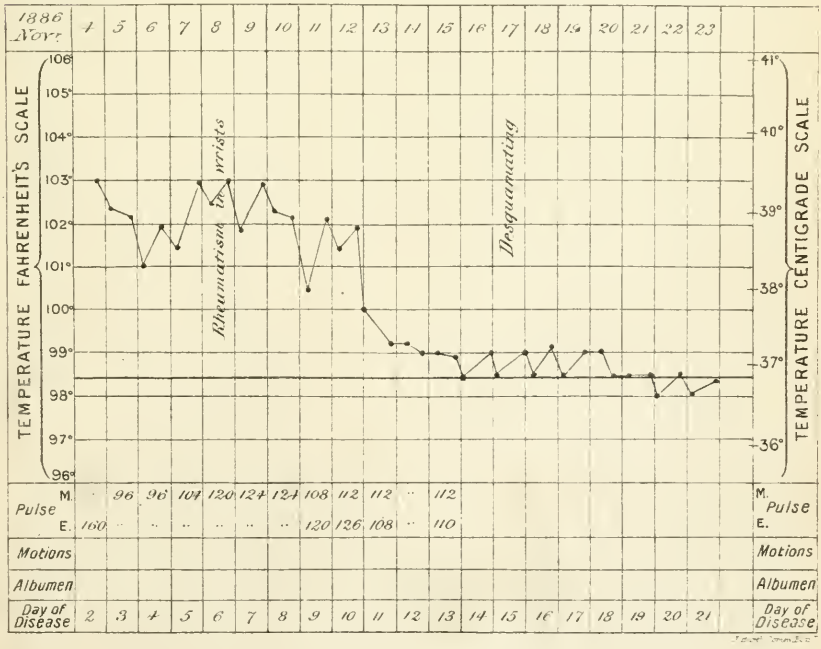
Name J. V. Age 7. Disease Scarlatina. Result Recovered

3.

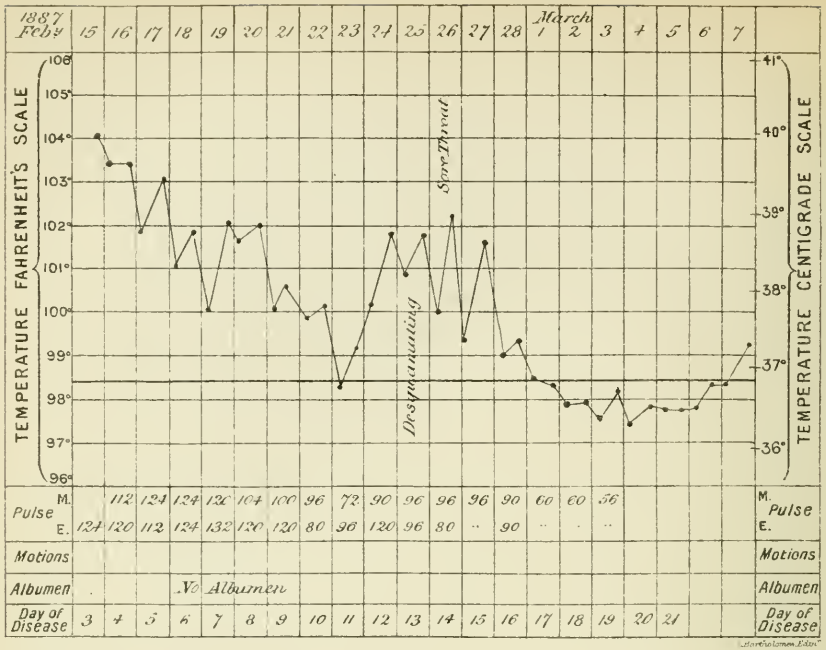


Name E. B. Age 5. Disease Scarlatina. Result Recovered.

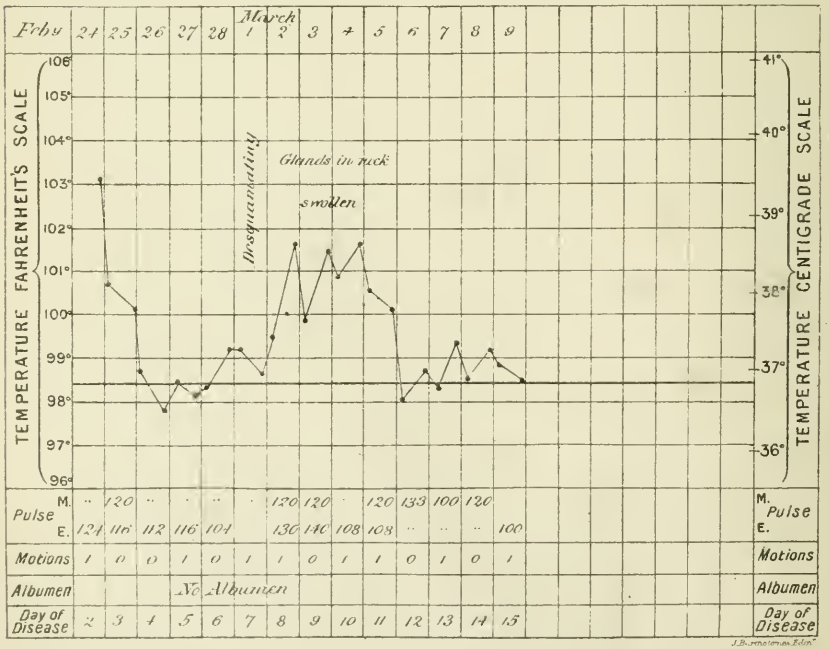
4.



5. Name Jessie Andrew. Age 18. Disease Scarlatina. Result Recovered.

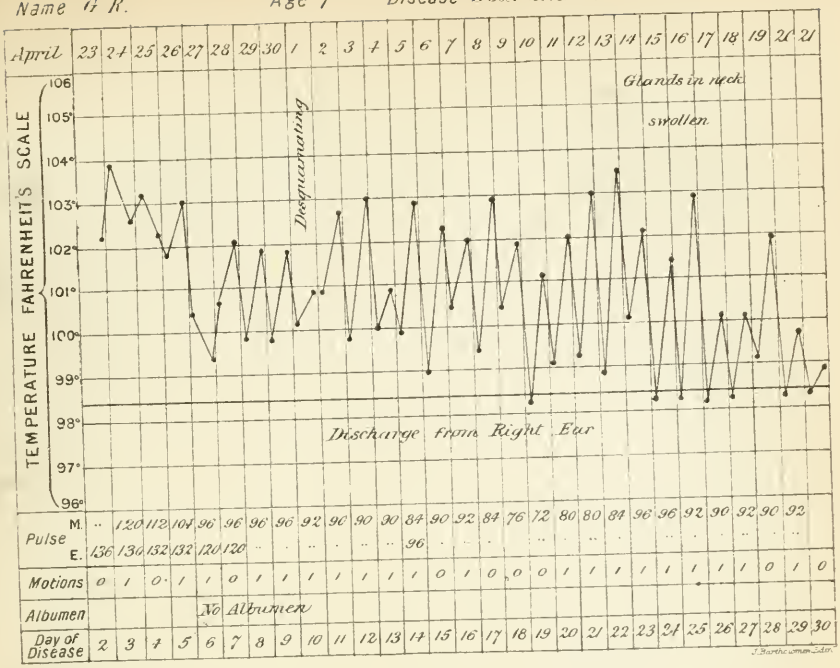


6. Name J. H. Age 6. Disease Scarlatina. Result Recovered.



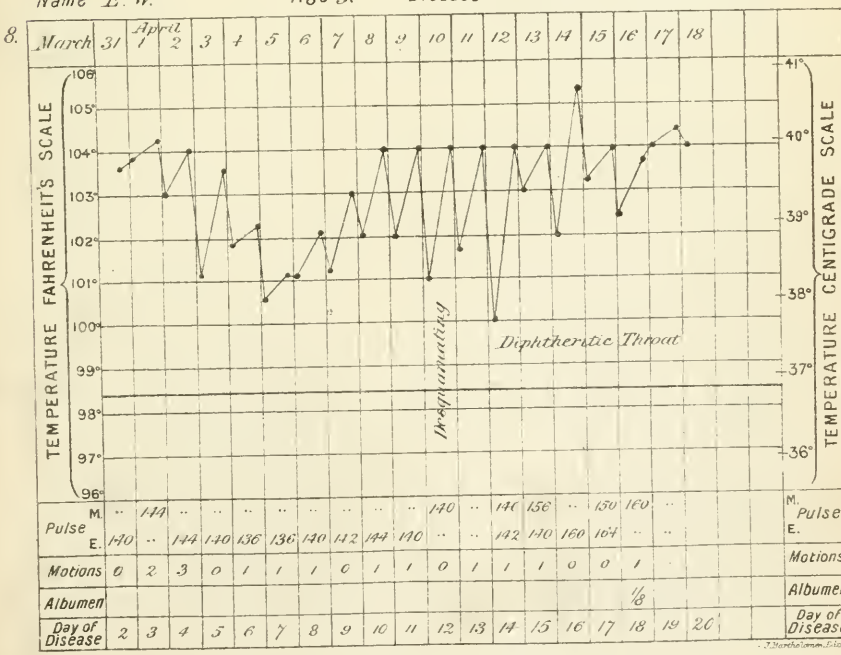
7.

Name *G. R.* Age *7* Disease *Scarlatina.* Result *Recovered.*



8.

Name *E. W.* Age *5.* Disease *Scarlatina* Result *Died.*



though certainly as a rule more copious in those in whom the eruption has been most vivid, it does not bear an absolute relation thereto. It is specially well marked, too, on parts where there has been little or no visible eruption, as on the palms and soles, and on the face.

With the separation of the cuticle the disease may come to an end, or certain complications may interfere with the steady progress of convalescence. These are not equally distributed throughout the same epidemic, and in different ones the relative proportions vary. In the order of frequency in the cases under consideration, there were thirteen cases of rheumatism, and a like number of swollen cervical glands; nephritis occurred in but seven instances, inflammation of the middle ear and otorrhœa in four, and secondary sore throat also in four. There is another troublesome complication which tends as much as any to keep up the temperature and prolong recovery, viz., naso-pharyngeal purulent catarrh. Of this we had some cases, but they have not been definitely noted, and so must be left out of consideration statistically.

Of the ordinary symptoms the eruption is the one which may be so transitory or so faint as to escape notice. Careful questioning has shown that in those old enough to observe the sequence, the primary sore throat always precedes, is not a subsequent symptom to, the rash.

Rheumatism is one of, perhaps the earliest of the sequelæ. In Chart No. 4 it will be seen that it influenced the temperature from the fifth day onwards. It affects the joints in the same manner as we are in habit of seeing in acute rheumatism of the conventional type, but the acid perspirations are absent or little marked. In one case pericarditis, and in another endocarditis occurred during its course.

Secondary sore throat is a term which I have applied to a parenchymatous tonsillitis which may terminate in abscess, or may disappear by absorption. Chart No. 5 illustrates its influence on the temperature, and the usual period of its occurrence.

Enlargement of the cervical glands may be single or multiple. When single and implicating the parotid or salivary gland, it may end in suppuration; when multiple, involution is the rule. It seems of septic origin, and to be due to absorption from the inflamed throat in the commencement of the disease. Chart 6 shows a rather acute example of this sequela; Chart 7, one in which there was also inflammation of the middle ear, and illustrates the way in which the temperature is kept up at a high though oscillating level for a long time.

In Chart 8 there is an instance of a rare and dangerous complication; in it diphtheria supervened early in the process, interrupted complete defervescence, delayed desquamation, and caused a fatal termination.

You, Mr President, having ruled that the discussion this even-

ing should be mainly directed to etiology and treatment, I must now briefly allude to these. Is there any mode of accounting for the peculiar course and sequelæ of scarlet fever? In only one way it seems to me. By the introduction into the system of a contagious material capable of setting up rapidly alterations of a fermentative character in the blood. There can be little doubt in any mind that this material is of the nature of an organism, which is certainly contained in desquamated cuticle in the later stages of exfoliation. An organism also which can indefinitely retain its vitality if kept dry, and which can also grow with great rapidity in milk. In this way only can we explain the fact admitted by Dr Klein, that one mode of communicating scarlet fever is from the desquamating flakes which have in some manner gained access to the milk imbibed. But while the contagium of enteric fever usually transmitted in a fluid medium seems capable of almost infinite diffusion, the dry scales from scarlet fever would go but a short way in spreading it, unless the particulate contagium contained in them could grow, and grow rapidly when sown in milk. The larger dose of the specific contagium introduced into the system in milk may account for the relatively increased proportion of cases of nephritis sometimes seen in a milk epidemic of scarlet fever. Having spent its force on the blood, and through it on the tissues, the organism enters the skin, and passes upwards through the various layers of the rete cells till cast off in the desquamating horny layer. There is no other possible way in which the cuticle could become contaminated. It would be somewhat surprising did the organism remain in a condition easy of detection in the blood after defervescence, and its disappearance therefrom as described by Dr Edington is less extraordinary by far than the way in which the embryo filaria vanishes and reappears. The rôle played by the organisms associated with the bacillus scarlatinæ in producing the sequelæ of scarlet fever has still to be wrought out. With the decadence of one organism may be combined the subsequent development of another. There is every reason for the assumption that acute rheumatism will one day be shown to be due to the presence and growth of an organism in the blood, and it is by no means unlikely that the same or a related one produces the rheumatism coincident with scarlet fever.

Of predisposing causes, age, the puerperal state, and the presence of an open wound, are the chief. Scarlet fever is a disease of youth, though but rarely of early infancy; and while not unknown in the elderly or even in the old, the susceptibility of the blood for the reception and increment of the virus diminishes with the advance of age. In this latter respect it contrasts with measles, to which those unprotected by having passed through a previous attack seem equally liable throughout life to its latest period. The condition of the blood after delivery

and surgical operations appears to be peculiarly adapted for the infection of scarlet fever, a statement again which does not hold good to anything like the same extent for measles. The organism of scarlet fever may thus be allied to certain septicæmic ones, as these also find a suitable nidus in the blood under similar circumstances. Examples are sufficiently common where individuals have long resisted repeated exposure to infection, and when their acclimatization might have been deemed complete, have acquired the malady.

There may at times be a difficulty in distinguishing between scarlet fever and some conditions which resemble it. In true surgical scarlet fever, according to Hoffa, who has recently discussed this question, the eruption takes its inception from the wound, or its vicinity, and spreads from thence. It must be borne in mind that not all cases in which a scarlet rash occurs in connexion with surgical operations or wounds are to be regarded as scarlet fever. Various erythematous eruptions are apt to appear under such circumstances, some of which are septicæmic, others are examples of drug eruptions due to the administration of cinchona compounds or derivatives, or of chloral, copaiba, or belladonna. In such cases the diagnosis is difficult, but the totality of symptoms is unlike those in scarlet fever. A scarlatiniform eruption has been described as occurring in association with diphtheria; of this I have seen one instance. All these imitations apparently differ in one particular: desquamation, which may be copious, usually begins very early, even before the subsidence of the eruption. It is rötheln, however, or German measles, which is most frequently mistaken for scarlet fever. One cause for this is that rötheln as a distinct member of the exanthemata has scarcely yet taken its proper place in the professional mind. In rötheln there is commonly some degree of coryza, of injection of the conjunctiva, and lachrymation, though by no means to the same extent as in ordinary measles. In scarlet fever injection of the conjunctiva only occurs in cases which present to a greater or less degree the characters of malignancy. In rötheln, too, the rash while on the first day it may be discrete and patchy like measles, may on the second day have become so uniform and punctate as to simulate that of scarlet fever. Perhaps of all the symptoms the condition and the behaviour of the tongue is that least apt to lead us astray when in doubt. In rötheln the tongue is thinly coated over the larger part of its area with a brownish fur, the sides alone being natural in appearance. When it cleans this fur melts away and leaves a normal surface. In scarlet fever the tongue on the second day is pretty thickly overlaid with white soddened epidermis, which separating from before backwards discloses a surface resembling raw beef, on which the normal epithelial covering is replaced rather slowly.

The goal of modern therapeutics is the attainment of accuracy.

We cannot make medicine an exact science, but our aim must be to approximate as nearly as possible to this. In treatment, therefore, we have largely abandoned the empiric measures employed in the past, the natural progress of disease has been subjected to minute scrutiny, and we know better when to hold our hand and when to interfere. If the charts which I have handed round show anything, it is that scarlet fever *per se* is self-limited, and runs, when uncomplicated, a pretty regular course. Till we knew something of the virus, anything like a rational treatment, as opposed to a mere guiding of the progress of the disease, was out of the question. If the bacillus scarlatinæ is really the organism which occasions the symptoms, it will now be possible to ascertain the influence of remedies in limiting its power. It would almost seem, however, that the rapidity with which the poison acts would negative any efforts to restrain it. Dr Dukes has suggested to us the administration of the biniodide of mercury, but to it there is one serious objection—the danger of mercurialism in those destined to become affected with nephritis. For the sore throat the saturated solution of boroglyceride in glycerine holds its own as the best local application, combined with weak mouth-washes of permanganate of potass. When the temperature attains a high level in uncomplicated cases, the best and safest antipyretic is a warm bath at a temperature of 100° to 101°. To give this in hospital we had large metal baths on wheels, which when filled could be run from ward to ward on the same floor, placed by the bedside, and the patient immersed therein and returned to bed without ever assuming the erect position. It is the primary rise when excessive that these baths do most to reduce; in secondary elevations due to complications they are less efficacious. Salicine proved most valuable in shortening the rheumatic seizures, and was regularly administered whenever the pain in the joints appeared. It is decidedly preferable to the salicylates in scarlet fever, as being less apt to cause gastric irritation. For the enlargement of the cervical glands, enveloping the neck in thick folds of cotton wool, painting with tincture of iodine, or the application of an iodide of potassium ointment made with lanoline, were used. The observations of Professor Guttmann would seem to show, however, that lanoline is not a better excipient of iodide of potassium than lard. Inflammation of the middle ear was treated by injecting warm boracic lotion, and when the acute stage had passed, by the daily use of Politzer's air bag, and with these measures the restoration of hearing was complete. Naso-pharyngeal purulent catarrh occurred solely in children; the treatment was therefore difficult, and consisted chiefly in the administration of quinine, and syringing the nose with boracic lotion. When renal dropsy appeared, acetate of potass was administered, an exclusively milk diet prescribed, and the hot pack employed with or without suitable doses of pilocarpine. The

separation of the thick epidermis of the palms and soles was hastened by rubbing in an ointment composed of resorcin 1 drachm, lanoline 6 drachms, and sesame oil 2 drachms. The loss of hair so frequent after scarlet fever was found to be obviated by the employment of this ointment as a pomade.

I have so recently placed before the profession a successful method of prophylaxis that it is unnecessary to say much about it now; but, as some seem to have misconstrued me, I would take this opportunity of stating that the claim for originality which I make is not the application of an antiseptic ointment, nor yet the use of baths in treatment, but the principle of anticipating the rise of the contagion through the skin, and neutralizing it as it does so by removing the loosening epidermis by baths and washing, and by the application at sufficiently frequent intervals, and from the very first, of an agent capable of killing the organism as it reaches the surface, and thus rendering the flakes which do separate innocuous. I would add that it is specially important that the head and hair be thoroughly washed with carbolic soap several times ere the patient is pronounced clear of infection, and is permitted to rejoin his friends. When this precaution is omitted, the scales dislodged from the scalp by scratching or in combing and brushing the hair are, I am sure, a not infrequent and an overlooked mode by which the disease is spread, either directly or through the medium of milk or other fluids, to which the scales from this region may readily gain access.

It only remains to be added that, from August to May inclusive, 500 cases of scarlet fever, in round numbers, were admitted into the City Hospital. Of these nearly one-half were taken in during February and March; the remainder were pretty equally distributed over the other months. Twenty-eight died, but of these ten were malignant cases, five dying within twenty-four, and five within forty-eight hours. If these are left out of account as being practically moribund on admission, the mortality in cases where time for treatment was allowed was only 3·6 per cent.

Mr Edington gave a demonstration of the bacillus, and related facts supplementary to those already published. He did not feel that he could say very much more in connexion with the bacillus scarlatina at this time, than to point out more in detail certain features with regard to its life history. In a previous paper, which appeared in the *British Medical Journal* of 11th June, the size of the organism was wrongly reported, as several had noticed on comparing the drawing of it with that of the bacillus arborescens. This bacillus, as seen in cover-glass preparations of fresh blood, measured 2_{μ} to 5_{μ} in length and $\cdot 4_{\mu}$ to $\cdot 5_{\mu}$ in thickness. Many microscopists had thought that it would be very easy to take blood and demonstrate it for themselves; but he pointed out that it was by no means an easy matter to get a case which was really not older than the third day, as the primary manifestations of the

disease were often not of such severity as to cause the patient much concern; and it frequently happened that when the medical man was called in the rash had assumed its retrogressive stage. Even if one was fortunate enough to get a case in the early stage, the bacilli might not be present in great numbers, and it might require care and patience to find them. Further, one might altogether fail to see any at all, and yet cultivations started with such blood might show them to have been present. If one resorted to cultivation experiments at a period about the third day, then one of three results would follow,—he might obtain a pure culture of the bacillus, or a mixed one of bacilli and micrococci, or he might find that the blood was quite sterile. From observations he had made he was convinced that there were two kinds of scarlet fever,—that in which the infection was due to the bacillus alone, and such would be found to run a purely typical course; and those in which the infection was due to the bacilli and micrococci. The latter case, as a rule, he had found to be associated with a very vivid rash, and followed by copious desquamation. In the case where nothing was obtained by cultivation, he had inferred that it was a case of bacillary infection, as whenever the micrococci gained access to the blood, one usually found them present, at least up to the end of the first week. Nevertheless, in such cases micrococci were found to appear a few days later. In cultivations one found that heat had a wonderful influence in stimulating the growth, *e.g.*, plate cultivations grown at 16° C. might be entirely liquefied in three days, and yet at a temperature of 19° C. the same result might obtain in thirty-six hours. Plate cultivations were characteristic: one found the liquefying colonies possessed a double contour, a central fairly circular ring, and an outer irregular one. It was found that that portion limited by the central ring was liquefied, while that portion between this and the extreme outer edge was not yet liquefied. And further, if we made, as he had here done, a cover-glass preparation of a whole colony, we saw that the bacilli in the outer zone were arranged in a definite order, and of an average size, $2{\cdot}5_{\gamma}$ in length and $\cdot 8_{\gamma}$ in thickness, while in the central liquid portion the size was irregular, and we might find an advance even to the leptothrix condition. This had been photographed by his friend Dr Francis Troup, and he was enabled, by Dr Foulis's kindness, to mount it and show it them by the oxy-hydrogen lantern. This organism was very capable of cultivation in more senses than one, for we found that successive cultures showed a more rapid growth-rate. With reference to this, he had grown it successfully in potatoes, as seen in the specimen, in which we found that growth began as a creamy, white pellicle, in the centre of which later a certain yellow coloration appeared, which spread outwards, the more central parts successively passing from this colour to varying shades of brown, until later it was entirely of a bread-crust colour; and with this deepening of colour may be noticed a progressive wrinkling, until

the whole growth later presented a tesselated appearance. On examining it at this stage it was found to consist almost exclusively of spores, which stained very slightly with a watery solution of methyline blue. He had grown such a cultivation from a rapidly-growing specimen, and on making from it again an inoculation into jelly, it was found that the growth-rate had not altered, or very slightly, thus differing from some organisms in which sporic formation seemed to check the rapidity of growth, and even to render cultivations made from it much slower than the previous cultures. With regard to control experiments on healthy skin, he had been conscious of their necessity; but not that they were absolutely so. He mentioned that 10 per cent. of the skin cases were those taken previous to the twenty-third day, and in no such case did bacilli appear. But the blood experiments were still better controlled. He reminded them that in each case the observation was made twice on the same day, with an interval of an hour or two intervening; and that while the bacilli occurred in the cases taken before the end of the third day, they never occurred in those later. Further, he mentioned that during the past two years he had taken blood and other discharges from the skin—somewhere between 150 and 200 times for Prof. Chiene—and while these had been conducted on the same lines, he had never before this ever seen such a bacillus; and, moreover, as cover-glasses that were taken of scarlatinal blood showed bacilli of such size and character as agreed with those afterward cultivated, he felt that he did not assume too much when he stated that these bacilli were inhabitants of the blood of those patients. He felt convinced that whatever be their nature, at all events their presence in scarlatinal blood would be amply corroborated in the near future.

Dr Foulis said — Sir Thomas Watson has truly said that "scarlet fever possesses the bad eminence of being the least uniform, the most unmanageable, and by far the most terrifying and deadly of all the exanthematous group." In speaking of the mortality of this disease, he says that "in eighteen years—from 1847 to 1864 inclusive—there died of scarlet fever in England and Wales 318,122 persons, giving an annual death-rate of 17,673. In the year 1869 it destroyed 27,641 lives, and in the year 1870 it destroyed 31,910 lives." Scarlet fever is the very type of what has always been, and still is, regarded as a contagious and infectious disease. The disease itself must be transmitted from the sick to the healthy by a specific poison. Experiments and observations prove that this specific poison is contained in the secretions of the skin and throat, and in the breath and in the discharges that escape from the nose and mouth in some cases. There is no evidence to show that the disease ever arises spontaneously. The specific poison always comes from the specific poison. It may be distributed among ourselves and our children in a hundred different ways. When sudden epidemics of this disease come upon

us, we must look to the air we breathe, to the liquids we drink, and to the food we eat, as the most likely vehicles by which the poison has been distributed. In the medical journals of the day we see recorded the histories of epidemics of typhoid fever, of diphtheria, and of scarlet fever; and it has been very frequently shown that these epidemics, in each case, have arisen from the use of some common article of food, such as water or milk. In Dr Aitken's *Science and Practice of Medicine*, seventh edition, vol. i. p. 513, there is recorded a very remarkable instance of scarlet fever infection by means of milk:—"A succession of cases of scarlet fever occurred in a college connected with the University of St Andrews several years ago. Servants were the first sufferers, then students. Some of the latter went away, carrying contagion with them, and occasioning fresh outbreaks; but the succession of cases in the college, where isolation and disinfection had been practised, was at first very perplexing. Scattered cases occurred in the town, also in families having no communication either with the college or with one another. At last it was found that the infection had been brought from a farm that supplied the several habitations with milk. This farm was a small one. The farmer's wife with one child visited a distant place where they were exposed to scarlet fever, and brought it home with them. The wife appears to have suffered very slightly. She nursed the child and milked the cows. A second child took the disease, and continued the infection. The boy who carried out the milk was next infected, and on him the fever was so slight that it never prevented him from accomplishing his daily round. On making out a list of the houses in which scarlet fever showed itself in St Andrews, and on asking the farmer for a list of those to which he sent milk, the two were found to be identical. With one exception the inmates of every house supplied with the milk had been attacked by the disease, and twenty-six cases and two deaths were directly traceable to this centre of contagion." In this case, it appears to me, there is not the slightest doubt that the milk was the vehicle by which the specific poison of scarlet fever was conveyed from a centre and distributed among a number of people living widely apart from each other.

As you all know, we have just had a very severe epidemic of scarlet fever in Edinburgh, which has brought misery and death into many houses in this city. During the twenty-eight days of February of this year 1887, 597 cases of scarlet fever were reported to Dr Littlejohn. Of these 386 cases occurred in the New Town, 145 cases in the Old Town, and 66 cases in the South Side. On the morning of the 10th of February I was called to a house in Ainslie Place to see three young persons down with scarlet fever of the worst type. These three children were all apparently in the best of health at 6 o'clock the night before—that is, on the evening of the 9th February. One of the young

persons was a page boy who lived on the kitchen flat of the house. The other sufferers were a little boy and girl, aged 7 and 5 respectively, who slept at the top of the house. When I saw them early on the morning of the 10th February, the three patients were in high fever, and delirious. Little or no rash was to be seen on any part of their bodies; the throats in each case were deep crimson. The children upstairs had supped on bread and milk at 8 o'clock on the night of the 9th, and the boy downstairs had also the same night supped on some of the same milk. The nurse who looked after the children next day was also found to be suffering from scarlet fever; and in a day or two the mother of the children, who would insist on nursing the latter, also took the disease in its worst form. The nurse and page boy were removed to the hospital and eventually recovered; the two children died; but the mother recovered after a great struggle. As the three young persons who were first struck down were apparently in the best of health at 6 o'clock on the evening of the 9th of February, it seemed as if they had been poisoned during the night, for they were so dangerously ill early on the morning of the 10th. After the most careful inquiry, it was found that bread and milk were the only articles of food in common between the three sufferers prior to the attack, and there was no history of their having been at any time exposed to scarlet fever infection. The milk they drank on the evening of the 9th February came from a milk-shop in the West End, which I shall call by the letter X. As soon as my visit to these children on the 10th was over, I had to hurry off to Douglas Crescent to see for the first time three grown-up young ladies with severe scarlet fever. In the afternoon I had to go back to the same house to see three grown-up young men, brothers of the ladies, who were now also down with scarlet fever. The whole six persons were in good health the previous day, and now on the 10th of February they were all down within a few hours of each other with bad scarlet fever. What had they in common? There was no history of exposure to scarlet fever. It is very rare indeed that one hears of six persons in the same family all sitting down to a breakfast of porridge and milk, but such was the case with this family. Each morning they all eat porridge and milk from a common dish. On inquiry where the milk came from, I found it was obtained from a milk-shop in the West End, quite close to the milk-shop X. I shall call this second milk-shop Y. During the next few days several other cases of scarlet fever came under my care, and in each case the patients were apparently in good health the day before the disease showed itself. In every case I found out where the milk supplied to my patients came from. Whenever I heard of a case of scarlet fever occurring among my friends and acquaintances, I made every inquiry as to the source of the milk used by them, and in this way I found out that quite forty cases had obtained milk from three milk-shops—X, Y, Z—all in

the west end of Edinburgh. I must now ask you to look carefully at the picture which I shall throw on the large screen before you by means of the oxy-hydrogen lantern. This picture is an enlarged photograph of the return of intimations of scarlet fever during the month of February. If you run your eye down the first two columns of the table, you will see that in the New Town during the first nine days of February there were but eighteen cases of scarlet fever reported to Dr Littlejohn ; but, all of a sudden, there is a remarkable rise in the number of cases reported daily.

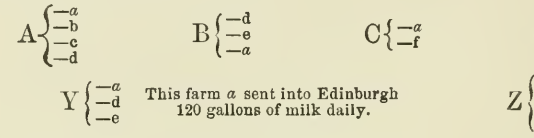
Return of Intimations of Scarlet Fever during the Month of February 1887.

Date.	New Town.	Old Town.	South Side.	Total.
1	2	8	4	14
2	4	9	2	15
3	4	3	2	9
4	2	7	...	9
5	2	3	2	7
6	...	6	1	7
7	1	4	1	6
8	1	3	4	8
9	2	9	...	11
10	8	8	3	19
11	12	...	...	12
12	33	4	2	39
13	31	4	...	35
14	51	2	3	56
15	52	3	3	58
16	42	12	1	55
17	13	6	1	20
18	17	9	3	29
19	10	9	3	22
20	7	3	2	12
21	3	6	5	14
22	12	5	2	19
23	12	...	6	18
24	22	4	1	27
25	12	3	3	18
26	14	7	10	31
27	11	4	...	15
28	6	4	2	12
Total,	386	145	66	597

On the	10th	there were	8	cases reported.
„	11th	„	12	„
„	12th	„	33	„
„	13th	„	31	„
„	14th	„	51	„
„	15th	„	52	„
„	16th	„	42	„
„	17th	„	13	„

From this table we learn that there were but 18 cases of scarlet fever in the New Town during the first nine days of February, and that in the next eight days—*i.e.*, from the 10th to the 17th inclusive —there were 242 cases. This rise in the number of cases is very remarkable, and there must have been a cause for it. Let us now look at another picture which I shall throw on the screen by means of the oxy-hydrogen lantern. It is a photograph of the city of Edinburgh, giving all the streets in their proper relation to each other. Whenever a case of scarlet fever is reported to Dr Littlejohn, he at once makes a little dot on the chart to indicate the locality in the street where that case is. In this way as dot after dot is marked on the chart, there is at last presented a most graphic picture of the epidemic as regards its locality and tendencies to spread. Such is the picture before you; but you will observe that some of the dots are coloured bright blue, while others are red. The blue dots represent cases of scarlet fever which were found on careful inquiry to have obtained milk from eleven milk-shops, to which I shall presently refer. There are nearly 200 blue dots on the chart before you. The red dots are also cases of scarlet fever, but in these cases it was not possible to ascertain from whence they got their milk supply. Some of them may have got, and probably did get, milk from the eleven milk-shops already mentioned. After very great trouble on the part of our officers of health, it was ascertained that quite 200 cases of scarlet fever did get their milk supply from the eleven milk-shops which are represented in the picture before you by little blue squares, each with a clear space in its centre. It is very interesting to notice that each little blue square representing a milk-shop is situated in the midst of a number of blue dots, or cases of scarlet fever, known to have got milk from them. This picture then shows, in a very striking manner, the relation, as regards locality, between the eleven milk-shops and at least 200 cases of scarlet fever. Now let us turn our attention to the milk-shops. What had they in common that could have given scarlet fever to at least 200 persons? Did they all get their milk from one common source? and was that common source infective? These eleven milk-shops were found to have got their milk supply from many different sources; but, strange to say, they were all receiving milk, in more or less quantities, directly and indirectly, from one farm situated a few miles out of Edin-

burgh. Let me make this clear to you. Let A, B, C, X, Y, Z represent certain milk-shops, and let *a*, b, c, d, e, f represent certain farms which sent to A, B, C, X, Y, Z daily a certain quantity of milk. By means of the following diagrams you will see that while A, B, C, X, Y, Z received milk from many different farms, they all received some milk from one farm in particular, viz., from the farm *a*.



What is there remarkable about the farm *a*, which is found to have supplied milk, directly and indirectly, to the eleven milk-shops? Now, gentlemen, I ask you to accept as truth what I am about to say concerning this farm *a*. I have obtained my information from a source that cannot be questioned. For obvious reasons I mention no names. Note the dates. On the 3rd of February the local doctor who attended at the farm was called to see a girl between 18 and 20 years of age. He found her suffering from a bad sore throat, and her body was covered with the rash of scarlet fever. This girl was the sister of the owner of the farm. On the 15th of February the same doctor was again called to see the owner of the farm. He was suffering from a sore throat, and had the scarlet fever rash on his body. What was the history of their infection? Thanks to the energy of Dr Littlejohn, it came out on inquiry that, at the end of January, somewhere about the 28th of the month, a young man, a brother to the girl and to the owner of the farm, had come from a neighbouring city to spend a day or two with them. This young man during the time he was at the farm was said to be in a state of desquamation after scarlet fever. There is no doubt that he was constantly in the society of his brother and sister at the end of January. It is said that he had some illness, which he thought was a cold and sore throat, at his own home, before he came to the farm to spend a day or two with his brother and sister. He left the farm before the 1st of February, and it was on the 3rd of February that his sister took ill with scarlet fever; and on the 15th his brother, the owner of the farm, was down with scarlet fever. At this time more than 100 gallons of milk daily were being sent into Edinburgh from this farm. It is but right to state that as soon as the girl was found to be suffering from scarlet fever she was isolated, and the same was the case with the brother who was taken ill on the 15th of the month. Now, the important question to answer is this, Had the milk which came to Edinburgh daily from that farm been in any way contaminated by either one or other of the three sufferers from scarlet fever? Whatever doubt may exist as to the affection from

which the visitor to the farm suffered, there is no doubt whatever as to the nature of the illness from which the sister and brother suffered. It was scarlet fever in their cases, without a shadow of a doubt; but they must have caught it from some one. It did not arise spontaneously in their cases. It is most likely that the desquamation from which the visitor was suffering was post-scarlatinal, and that he infected his brother and sister. The doctor did not see the cases of scarlet fever *until* the sore throat and rash were out and well marked in each case. What were these two patients doing, say, during the twenty-four or forty-eight hours *before* the well-marked symptoms of scarlet fever appeared on them? Were they then in any way employed in connexion with the supply of milk to Edinburgh? This and many other questions one would like to have answered in the interest of truth; but it is quite impossible now to get such answers, and it is no use guessing. What we do know is this, that in the New Town of Edinburgh, from the 10th to the 17th of February, there arose a great epidemic of scarlet fever; and that of the cases at least 200 were found to have obtained their milk supply from eleven milk-shops, all of which were at this time receiving milk from a farm on which there were two well-marked cases of scarlet fever. Though these eleven milk-shops got their milk from many different sources, they all had in common some milk from this farm; and they were proved to have had nothing else in common. Believing in the terribly infectious nature of scarlet fever, I can come to no other conclusion than that the milk from this farm was the vehicle by which the infection was distributed to the milk-shops, and by the latter to the sufferers. Now, gentlemen, it is right to mention a fact which we, as medical men striving to check disease, should know. It was not until early in March that the facts I have stated were known to our officers of health. They certainly were not to blame. Had it not been for the energy of Dr Littlejohn and others, we should probably never have known that there were cases of scarlet fever at the farm which sent so much milk daily into Edinburgh. The law compels us who live within the city boundaries to intimate to Dr Littlejohn within twenty-four hours the existence of every case of infectious disease that comes under our care. This law, however, does not apply to medical men who live outside the city boundaries. They are not compelled to give any notice of the existence of infectious diseases under their care to the officers of health, who alone have the power to insist on immediate and complete isolation of such infectious cases. Suppose, now, that Dr Littlejohn had been made aware of the existence of scarlet fever on this farm on the 3rd of February, he would undoubtedly at once have stopped the supply of milk from that farm. Instead of this having been done, the milk was allowed to go into Edinburgh daily. Notice that on the 10th of the month the great rise in the number of cases began. All this time the milk from the farm was

coming into Edinburgh. *Infection must have existed on the farm, because on the 15th of the month the second case of scarlet fever occurred there.* The milk still came into Edinburgh from the farm, but by this time both patients were in bed, and completely isolated and protected from all risk of contaminating the milk. The return of cases shows that after the 16th of the month there was no further rise of cases, but that there was a gradual falling off in the number. By this time the disease was well established in Edinburgh, and every one knows that it may spread in a hundred different ways after desquamation has begun.

The same thing may, and will, occur again, unless we medical men rise up and agitate for the immediate extension of the law of compulsory notification of infectious disease. All medical men, both within the city boundaries and without the city boundaries, in my opinion, should be compelled to intimate to the local authorities the existence of infectious disease at once,—not a moment should be lost in bringing the existence of such cases under the notice of our health officers. In this way only can infectious disease be properly isolated and checked at the beginning. Ignorance and self-interests are important factors in hiding the causes of disease; but where educated and thoughtful medical men know of the existence of such diseases, nothing is left to them but to rush immediately to the help of those who have the power to check the spread of the disease at its beginning. Our knowledge is our power. The time has long gone by when in our ignorance we used to say that Providence sends us these dreadful diseases for our good! No, the time for such belief has gone. Let us work and find out the causes of disease, and then happily we shall be convinced that all these infectious diseases are preventible. If you had witnessed, as I have lately, the awful misery and suffering in the houses where this dire disease of scarlet fever has made havoc among children, you would agree with me that it is our highest duty to search for its beginning, and then to rise up and act with decision, and without hesitation to crush out of existence this monster of disease and death. Do not let the lessons we have learned from the study of this epidemic of scarlet fever pass away unheeded. I call upon you, Mr President, to lead us in an agitation for the immediate extension of the Act for the notification of infectious disease to beyond the city boundaries, in such a way that every medical man shall be compelled to notify immediately to the local authorities the existence of infectious disease under his care.

Mr A. W. Hare spoke of the relation, now all but universally accepted as existing, which milk supply bears to epidemics of scarlatina. The chief evidence of this relation up to the year 1881 had been collected and epitomized by Mr Ernest Hart in the *Transactions of the International Congress*, London, 1881; and the proof therein supplied was, he thought, entirely conclusive. It

had been established that the scarlatinal poison might reach the milk from the atmosphere, by transference of infective particles in suspension in it; by water, used either for cleansing or diluting purposes; from fomites; or from direct contact with the human subject in the manner discussed by Dr Foulis. It remained, however, to inquire whether there were no other means of contamination of milk—whether, in fact, the virus might not be autogenetic as regards the milk, due to the occurrence of some special bovine disease in the animals supplying it. Mr Hare then gave a *résumé* of the papers by Drs Power, Cameron, and Klein in the *Report of the Medical Officer to the Local Government Board for* 1885. The conclusion to which these researches distinctly pointed was that the scarlatinal virus is in some cases communicated to milk from disease in the animals which yield it. Through the kindness of Dr Klein, he was able to show to the Society cultivations of Klein's diplococcus under varying conditions of growth, and derived from various sources. The strong chain of evidence referred to has been recently completed by Dr Klein's discovery in the blood of patients suffering from scarlatina of the same organism he had previously described as the *materies morbi* in other situations where it had given rise to infection. Returning now to Mr Hart's cases, he found that out of the fifteen epidemics of milk-scarlatina summarized by him, in four there was absolutely no evidence of contamination of the milk from human sources, while in four of the remaining eleven cases where contamination had been so traced the evidence was so flimsy as in no way to amount to proof. A study of the records of such epidemics would thus lead one to look for other sources of contamination than those usually accredited; and to the speaker's mind Dr Klein's investigations and conclusions very satisfactorily filled the hiatus thus left in the etiology of the disease. In regard to the question of disinfecting milk, heat was undoubtedly the only available means; but it must be remembered that to sterilize milk was extremely difficult, on account of its peculiar physical properties, and for this reason it was hardly ever used as a culture-medium by the bacteriologist, though in other respects it was pre-eminently fitted for such purposes. This difficulty might be obviated if the scarlatinal virus was really destroyed at as low a temperature as that described by Dr Klein; but it appeared to him that all such measures were inefficient palliatives; the root of all epidemics due to milk lay in the dairies that supply it, and it is only at the root that such outbreaks could be properly and radically treated. They must look more to stringent dairy inspection, and, if possible, a strict system of dairy licensing, for relief than to any system of disinfecting dairy products.

Bailie Russell said that, after reading the report of the Hendon investigation, he did not consider it was sufficient to prove the existence of a bovine scarlatina capable of being communicated to man. In the course of the recent epidemic in Edinburgh he had

seen several cases where scarlatina was not recognised, even by medical scrutiny, until desquamation revealed the true nature of the slight illness. The existence of such cases constituted a source of fallacy in investigations like that at Hendon, and rendered it improbable that scarlet fever could ever be completely stamped out. In searching for the cause of fever in the late outburst due to milk, they met the difficulty that scarlet fever had been for some time prevalent in the town, and that the milksellers did not all give a full statement of their sources of supply when questioned by Dr Littlejohn's inspectors. That it was not due to some local influence or to contact in school was shown by the simultaneous invasion of families in a large working-class district containing several schools, and of families scattered over wealthy districts in the town. The children of the poor and rich did not attend the same schools, but they had this in common, that milk from one dairy farm was sold to the milk-shops which supplied all these families. Another interesting proof that the outburst was not due to schools was seen in the mode of invasion of the scholars attending North Merchiston Board School. These scholars came from two very distinct districts, situated on opposite sides of the Canal. For some time no cases occurred among those living on the south side of the Canal, while there were many on the north side, where the beforementioned milk was distributed. At a later period cases began to appear in a sporadic manner on the south side, being evidently due to infection at school or in some other common way. The clinical features of the invasion, as reported by Dr Foulis in a letter to the Convener of the Public Health Committee, strongly negatived the idea of infection except through some article of diet from a common source. In each of two families half a mile apart the unprotected milk consumers (nearly half a dozen in each) were attacked *simultaneously*, and it was found that their respective milk-shops received milk from the farm where scarlet fever was subsequently discovered. The other positive evidence consisted chiefly of the facts that not a single case of scarlet fever was reported during the first week of February among the customers of the eleven milk-shops wholly or partially supplied with milk from the dairy farm in question ; that these customers suddenly began to be attacked during the second week in a ratio much greater than the customers of other milk-shops to which this milk was not supplied; that wherever farms supplied other shops in addition to the eleven sellers of the special milk no undue number of fever cases occurred among the customers. At the farm the history of events was as follows, so far as could be ascertained. A relative in Glasgow suffered from supposed cold and severe sore throat, and came to visit his friends in the latter part of January. He is said by one observer to have been desquamating. At this time scarlet fever was unknown in the neighbourhood, but some days later, towards the beginning of February, the local medical man was called in,

and recognised scarlet fever in the family. The cow-houses and milk-houses were situated at a little distance from the dwelling-house where the farmer's family and attendants on the cows lived. Then the outburst in Edinburgh began, and two children of one of the outdoor farm-servants who had milk from the farmer became affected. The farmer denied that his milk was the cause of fever in Edinburgh, and wrote to threaten the speaker with an action for what he had said in public upon the matter. It was to be remarked that the existence of scarlet fever at this farm was unknown to the local authority of the parish for nearly three weeks, *i.e.*, until the outbreak in Edinburgh had taken place; and it is doubtful whether, even had it been known, their officials would have been allowed to communicate the fact to the Edinburgh authorities. Midlothian at that time had no fever hospital. The lessons of this epidemic for the people of Edinburgh will not be thrown away if they cause an agitation for reporting fever cases in the country, and other amendments on the law and administrative machinery for protecting the public health.

Dr Affleck remarked that the object aimed at in a discussion of this kind should be at once the discovery of truth, and the exposure and elimination of those "false facts," as Cullen termed them, which did so much to retard real progress in medicine. Referring to the recent Edinburgh researches, he gladly acknowledged in common with his brethren the excellent work done by Drs Jamieson and Edington, but thought that it could only be regarded as a very preliminary contribution to the settlement of the biological problems included in the etiology of scarlet fever. He regretted the absence of controlling or parallel experiments with healthy skin, or skin desquamating from other causes, and thought that a lengthened period of time must be given to this research to produce results upon which much could be founded. The experimental part of the work had failed to carry conviction to his mind that the bacillus found was that of scarlet fever, and the obtaining, by its inoculation into animals, an erythematous rash followed by desquamation, did not afford satisfactory proof that this morbid appex was that of scarlet fever. He regarded it as extraordinary that in none of the researches here or elsewhere had the attempt apparently been made to introduce the organism into the human body, which might have settled the problem in a way which could admit of little doubt. There were, however, other points in the etiology of the disease upon which he felt more at liberty to express an opinion. He feared that very loose notions prevailed, not merely among the public, but also in the profession, as regards the infection of scarlet fever—a common remark being that this malady is not very infectious at first, and that it is chiefly when the desquamating period has been reached that the danger arises. While admitting the great influence of desquamation in spreading the disease, Dr Affleck held strongly that

scarlet fever was highly contagious from the very outset. He had had many sad lessons to this effect both during the years he had had charge of the Royal Infirmary fever wards and otherwise, and he thought medical men ought to point this out more emphatically to their patients, and be more strict in acting upon it themselves in practice, by the prompt isolation of every case on its first appearance. It was no argument to point, as was sometimes done, to the experience of nurses who, while waiting upon scarlet fever cases, had not contracted the disease till desquamation had begun, inasmuch as it was to be remembered that the nurse had all along been imbibing the poison, and that it required a certain amount to be introduced to work its effects. The analogy of the soil and seed held here, and the personal factor also was always to be reckoned with. In some the soil was barren, in others it brought forth an hundredfold, the very minimum exposure, as he had many a time proved not only in this but in other fevers, being sufficient in some persons to beget the disease in its most appalling and fatal form. Referring to the recent epidemic in Edinburgh, Dr Affleck could not take the strong position held by Dr Foulis and Dr Russell, that there could be no doubt it was in certain localities clearly traceable to milk contamination. For although, no doubt, the circumstances as narrated were highly suspicious, yet scientific proof was lacking. In the first place, it was to be remembered that an epidemic had been for some time in progress, and from what they had often observed in epidemic visitations of any disease, the malady does not all at once fall upon a community, but steals in here and there for a time, and then suddenly bursts with fury upon some special locality or throughout the whole district. So it was in past times both with small-pox and cholera. Further, even admitting the existence of a case or cases of scarlet fever at the dairy farm, it did not necessarily follow that the disease must be conveyed to the milk; and the inquiry, to have been scientifically complete, should have extended to all the dairy farms which sent milk into Edinburgh. On the whole subject of milk infected in scarlet fever, Dr Affleck thought the evidence was of inferior strength to that which existed in the case of enteric fever, where in numerous instances an epidemic outbreak of typhoid in a previously unaffected place had been clearly traced to the dairy, where the fever was prevailing, and where milk or water contamination was perfectly proved. Finally, upon the point of treatment, Dr Affleck thought that Dr Jamieson's plan, while it had apparently certain points to recommend it, had much more to be said against it. He could not admit that any system of antiseptic inunction and throat brushing could avail to prevent the spread of a disease that was being eliminated by probably every available channel, and to say nothing of the questionable practice of interfering with the important function of skin-elimination, how could the application of the

most approved antiseptic to the throat, or rather only a part of the throat, have more than a transient effect, when we remembered that by the secretions the whole application would be washed off in the course of a minute or two, leaving the parts very much as they were. It would require much stronger evidence than had yet been forthcoming to satisfy him that it would be safe by this inunctive plan to allow the fever patient to mix indiscriminately with the healthy members of a family. All that he (Dr A.) had seen of this disease, had more clearly proved to him that the true lines of treatment were prompt and complete isolation, prolonged care and watchfulness for complications, and a light and strictly guarded diet, together with systematic measures of disinfection,—in short, the plan pursued, though not perhaps with equal strictness, by every enlightened practitioner.

Dr Hunter (Linlithgow), said that, whatever the organism was, it was evidently markedly affected by environment. It was an observed fact that the virus of scarlatina, when brought in contact with a puerperal woman, afforded different manifestations, according as the contagion reached the patient directly by the genital passages (when it gave rise to metritis, parametritis, perimetritis, and puerperal septicæmia), or in the ordinary way, when scarlatina was produced. Dr Jamieson's treatment of the throat was carried out in young children with difficulty. As to enlarged cervical glands, treatment should be directed to the naso-pharyngeal mucous membranes, from whose surface the absorption occurred which gave rise to the adenitis. The direct treatment of the adenitis, as suggested by Dr Jamieson, was not sufficiently thorough-going. It could be merely palliative.

Dr Andrew Smart referred to the Report on the Rinderpest published by him many years ago, and remarked that, in reading the recent papers of Dr Klein and Dr Power, he had been struck with the resemblance existing between the lesions found in the Hendon cows and those discovered by him in the Edinburgh cattle. The symptoms and pathological conditions were almost identical. He was inclined to think that the Hendon cows were really sporadic cases of the same disease which had proved fatal to many of our cattle at the time to which he referred. In his *interim* reports to the Edinburgh Town Council twenty years ago, Dr Smart had suggested the striking resemblance of the Rinderpest in all its destructive characters to human scarlatina.

Dr W. Russell said that as the subject had been so fully gone into by previous speakers, he would not further refer to the etiology of scarlet fever, than to say that they were not yet in a position to form any definite opinion; but he hoped, for the credit of the school, Dr Edington's contentions would be found to be correct. He would confine himself to making a few remarks on Dr Jamieson's paper. With reference to the use of inunctions for destroying the infectiveness of the desquamating cuticle, he had

used carbolic oil with this object for so long, that he had forgotten from whom he learnt it, and he knew many practitioners who used it. He was not, however, prepared to rely on inunction alone, but insisted on the most complete isolation, if isolation was attempted at all. With reference to the treatment of the sequelæ, his experience entirely agreed with Dr Hunter's, that the best and proper method of preventing or restraining the enlargement of the cervical glands was by applications to the fauces. He expressed his belief in the treatment of nephritis by active purgation, but he thought that of recent years he had seen less nephritis after scarlet fever than he used to, and that this was probably due to using the hot bath or pack in treatment. Dr Jamieson had suggested that nephritis was common when infection was conveyed by milk; if this were so, and if there was any truth in what they had heard that night, nephritis ought to have been of the common sequelæ in the late epidemic, but it was not in accordance with his experience of that epidemic that this was the case. Dr Jamieson had not referred to malignant scarlet fever, nor thrown out any indications or suggestions as to the treatment of this form, which was one of the most appalling things met with in practice.

Dr Allan Jamieson said that he would only allude to one or two points of the many interesting ones which had been raised. Dr Affleck had complained that no inoculation experiments had been made on man. When in the course of their investigation they had reached the stage of producing a rash and desquamation in rabbits, he had solicited volunteers from the students attending his clinique at the Royal Infirmary, and four had indicated their willingness to be inoculated. Before doing so, however, he had held a consultation with Professor Chiene and Dr Edington, and it was determined to pursue experiments on animals still further first. Immediately after this the first calf died, and of course all idea of inoculating the bacillus was at once abandoned. As regards prophylaxis, nothing was further from his intention than any encouragement to carelessness with respect to infection. The method was meant as a valuable adjunct to isolation where that was possible; but there were many cases where removal to an hospital was impracticable, and isolation at home, from the restricted nature of the accommodation, out of the question; for such the measures recommended would be found of the greatest value.

In consideration of the lateness of the hour, it was agreed, on the motion of the junior secretary, to adjourn the debate.

After remarks by the *President*, the meeting was adjourned.

Special Adjourned Meeting.—October 5, 1887.

Professor GRAINGER STEWART, *President, in the Chair.*

Mr Hare read the second report.

Since submitting to the Society the preliminary report, your committee have further prosecuted the investigation of facts relating to Mr Edington's paper on the nature of the contagium of scarlet fever.

The committee have now collected a considerable amount of evidence bearing on the several points under consideration. While, however, the facts before them are of considerable importance, they do not as yet feel themselves in a position to present a final report.

The following is a summary of their work:—

1. *Microscopical Examination of the Blood.*—Blood from ten typical cases of scarlatina on or before the third day of the disease, was examined microscopically for the presence of micro-organisms. In this part of the investigations forty-four specimens were examined under the microscope, with the result that in the blood of one patient bacilli and micrococci were found, both in small numbers; in that of two patients bacilli and micrococci were found, the former in very small, the latter in moderate numbers; in that of four cases micrococci alone were found; and in the remaining three cases no micro-organisms could be detected.

2. *Cultivation Experiments with the Blood.*—The blood was taken from nine typical cases of the disease on or before the third day. In four of these cases bacilli and micrococci appeared in the culture material, in two micrococci alone, and in three a negative result was obtained. It is worthy of notice that the cases in which the microscopic observation failed to discover micro-organisms only in one instance corresponded with those in which cultivation experiments failed. So that taking the nineteen observations together, evidence of the existence of organisms was obtained in thirteen; or, taking the series of ten cases to which those observations refer, evidence of a positive nature was obtained in nine of them.

The committee are not yet in a position to describe all these forms of microbes, but they are at present engaged in the investigation of the nature of the several organisms.

3. *Cultivation Experiments with Desquamation.*—One typical case was examined, with negative results.

4. *Several Experiments* were performed on calves with the "bacillus scarlatinæ," supplied by Mr Edington. These consisted of an inoculation experiment, by subcutaneous injection and scarification, with this material; a feeding experiment with the same; and a combined feeding and inoculation experiment. In each case the result was negative.

5. *In conjunction with Professor M'Fadyean* of the Royal Dick Veterinary College, to whom the thanks of the committee are due, three experiments were performed, in which it was attempted to induce scarlatina in calves by injecting scarlatinal blood in two cases, and by feeding with desquamated scales in the third. The materials were from typical cases of the disease. These experiments also gave negative results.

6. *The committee feel warranted* in expressing their belief that the "bacillus scarlatinæ" of Edington is not, as suggested by some observers, the bacillus in eczema (Smith), the bacillus butyris, nor the bacillus subtilis. They have, however, not yet been able to compare it with certain other forms of bacillus which it is said to resemble,—*e.g.*, the bacillus epidermidis, the bacillus of acute rheumatism (Mantle), and the bacillus found in the skin in erythema marginatum (Mantle).

7. *They believe that they have definitely ascertained the fact* that the micro-organism described by Mr Edington under the name of the "streptococcus rubiginosus," is morphologically identical with the "streptococcus scarlatinæ" of Klein. The value of this identification is emphasized by the facts as to inoculation experiments described by Mr Edington in a letter appended to this report.

In conclusion, your committee beg to state—(1.) That they find that an organism resembling that described by Mr Edington as "bacillus scarlatinæ" is, in a certain proportion of cases, present in the blood taken with due precautions from scarlatinal patients. (2.) That they believe that the streptococcus scarlatinæ of Klein is identical with the streptococcus rubiginosus of Edington. (3.) That they have as yet failed to corroborate the conclusions arrived at by Mr Edington as to the pathogenetic value of the "bacillus scarlatinæ."

The committee desire to obtain the sanction of the Society to continue the investigation, particularly as to—

a. The morphology of the several organisms.

b. The susceptibility of various animals to infection with scarlatinal blood and with the various organisms, their investigations so far having led them to doubt whether calves are susceptible to the action of the human scarlatinal poison.

They have therefore to ask the sanction of the Society for such further expenditure as this fresh line of experimentation may demand.

Subjoined is a letter with enclosure, which has been received from Mr Edington, to which reference was made in the report, and which the committee deem of such importance as to place before the Society *in extenso*.

(Signed) T. Grainger Stewart, *Convener.*
James Carmichael.
G. Sims Woodhead.
Rob. W. Philip.
Arthur W. Hare.

44 Great King Street, Edinburgh,
3rd October 1887.

Professor Grainger Stewart, M.D., Convener of Medico-Chirurgical Society's Committee for the Investigation of the "Bacillus Scarlatinæ," etc.

Dear Sir,—I herewith send you a letter received by me from Dr Roswell Park, M.D., Professor of Surgery, Med. Dept. University of Buffalo, together with microscopic preparations which accompanied it. The letter fully explains what they purport to be. Having carefully examined them, I am unable to see any distinctive difference in them from the bacillus which I have described; but I have intimated to him that these preparations of themselves are not sufficient to enable me to say that they are the same. You will, however, note that he states that cultivations in different media correspond with those described by me.

In response to your question as to my experimentation on man with scarlatinal material, I beg to say that having inoculated a tube of gelatine with scarlatinal blood of the fourth day, I was successful after thirty-six hours' incubation in getting a profuse cultivation of streptococcus rubiginosus and diplococcus scarlatinæ sanguinis, and having emptied half of it into some fresh milk, it was drunk by my brother. Three days later, his blood on being examined showed micrococci, which being cultivated, showed that the streptococcus rubiginosus was present. His temperature was carefully taken morning and evening for fourteen days, but never showed the slightest pyrexial rise. I might also state that he never has had scarlet fever, and from this fact I doubt very much the pathogenicity of this organism in man.—I am, dear Sir, yours respectfully,

(Signed) Alexander Edington.

Buffalo, 24*th August* 1887.

Alexander Edington, M.B., C.M.,

Dear Sir,—We have been very much interested in your papers on the bacillus scarlatinæ in Nos. 1380 and 1388 of the *British Medical Journal*, especially so because we have for the past fifteen months been cultivating a bacillus which seems to correspond with the one described by you. It was taken from the blood of a case of scarlatina, where the eruption was well marked, by puncturing the skin under due antiseptic precautions. It has been cultivated on gelatine, agar-agar, and potato with the same characteristic growth detailed in your articles, and has seemed to be a comparatively pure culture from the start. Just now we have a beautiful potato cultivation which is undergoing the changes of colour you described. The mounted specimens accompanying this letter were taken from it on the 5th and 6th days respectively.

We have had no time to make inoculation experiments or to study the bacillus carefully, and are anxious to know whether it really corresponds to the bacillus found by you.—Respectfully yours,

(Signed) Roswell Park, M.D.,

Professor Surg. Med. Dept., University of Buffalo,
per T. N.

The President said that he thought every member present must have been interested in some at least of the statements of this interim report, and in particular, with the announcement that the committee had satisfied itself of the morphological identity of one of the organisms described by Mr Edington with that which Dr Klein regarded as the essential cause of the disease. The mere fact that the streptococcus scarlatinæ of Klein is really morphologically identical with the streptococcus rubiginosus of Edington is important. If it should turn out that these two organisms are in other respects the same, the interest would be increased, especially seeing that Mr Edington's letter appended to the report gives an account of an experiment performed with this organism upon the human subject. The subject selected was one who had not had scarlet fever. A cultivation of the streptococcus rubiginosus was introduced into the stomach. Corresponding development of micro-organisms occurred in the blood, but the patient never got out of health, and there was no rise of temperature. No single experiment could be more conclusive than this, and its importance would be manifest if the committee's conclusion was found to be correct. The committee had found the bacillus scarlatinæ of Edington in the blood of several patients, although in others they had failed to find it. This part of the research had been carried out with great care by Drs Hare and Woodhead. The failure of the inoculation experiments was disappointing, and they desired to make further experiments, and he hoped that the Society would see its way to prolong the existence of the committee, and to authorize somewhat larger expenditure.

At the private business meeting of the Society, it was agreed to continue the committee, with powers to make further experiments.

Dr James Ritchie said there are few more trying positions in which a physician may be placed than at the bedside of a patient who but a few hours before seemed to be in perfect health, but who has been absolutely felled by the poison of scarlatina, whose quick feeble pulse, convulsions and unconsciousness, indicate a speedily fatal termination. Not less trying is it when the symptoms to the practised eye indicate a termination less speedy, but none the less sure. The discovery of the poison of scarlatina would enable us to discern a streak of light in the dark horizon of prognosis in such cases. But before the discovery of the micro-organism of scarlatina can be of any practical use, there remains much to be done. The bacteriologists must tell us in what media

it flourishes, whether acid, neutral, or alkaline; does it produce a ptomaine, and if so, what is its antidote? what renders it more virulent, and what less so? This question of virulence is of the highest importance. A very able physician told me some time ago that he used to ascribe fatal results in scarlatina to injudicious interference on the part of the physician in attendance. My friend had his error rudely discovered to him; but in holding the above opinion he sinned in good company, for Bretonneau, who during 23 years never saw a fatal case, when in 1824 there set in a virulent epidemic, he at first blamed the treatment of his colleagues till he was himself brought face to face with the disease, when he found that he had to deal with one as fatal as the plague, typhus, and cholera. Probably we all remember Sydenham's description of scarlatina, "Hoc morbi nomen, vix enim altius assurgit." Graves, during the first four years of the century, found it very fatal; for the next twenty-seven years it was hardly ever fatal, but during the following year, 1832, he found it more deadly than previous epidemics of typhus and of cholera. Why this variability in different epidemics? But not only does its virulence vary in different epidemics, but during the same epidemic in different parts of the same locality. I believe there are at least three factors,—1. The virulence of the poison; 2. The constitution of the individual, or we might say of the family; 3. The constitution of the period in relation to disease. Of the last of these I know nothing practically. Sir R. Christison used in his lectures to refer to it. Autenrieth writes of it as the "constitutio morborum stationaria." The peculiarity referred to is the tendency at certain periods for all acute diseases to be followed or accompanied by great prostration; at another time to be characterized by the amount of inflammatory reaction; while again, disorders of the liver and of the gastro-intestinal canal are the common features. Regarding the second factor, there have been great differences of opinion; my own experience leads me to conclude that strumous children, and those with a syphilitic taint, take scarlatina severely, and that those with enlarged tonsils are most liable to some of the complications. As to the cause of the virulence of the poison we have not sufficient knowledge. As milk is so favourable a nidus for the development of micro-organisms, we might, *a priori*, expect milk epidemics to be severe. This is not, however, the invariable experience. But we desire our friends the bacteriologists to do more than to give us information regarding the micro-organism which causes scarlet fever. Why is it that there is so great a variability, not only in the virulence of the disease, but so great a variety of type? Why in some, after the fever has declined, does a severe form of sore throat set in; and why, after this severe sore throat, is one patient affected with glandular enlargements, and another with a brawny phlegmonous collar? Why in some does a mild rheumatism supervene, and in others a

severe affection of pericardium and joints? We wish the bacteriologists to tell us whether the micro-organism of scarlatina, after having flourished, multiplied, and compassed its own destruction, does not prepare the way for some other kind or kinds of micro-organisms, and if so, what is the life history of these; or does it only act by enfeebling the constitution of the patient, and so diminishing its power of resistance to the inroads of some other form of microbe? For such information we wait patiently; meantime let us endeavour, by comparing notes, by an interchange of dearly bought experience, to further our knowledge of the treatment of the disease. Those *rapid malignant* cases, which, when first seen are moribund, we can never hope to benefit—these lead us to think well of prevention; but in those in which the *fatal result is delayed*, I am hopeful that some day we may be able successfully to combat the poison. The direction in which we ought to work is to endeavour to find a means of destroying the vitality of the organism, and to obtain an antidote to the ptomaine, which probably acts by paralyzing the sympathetic system. The biniodide of mercury has recently been used in this disease as a germicide. Dr Illingworth, who has devoted some attention to this remedy, may this evening give his experience of its powers. Some years ago, Dr Brakenridge, influenced by the experiments of Polli and of Sansom, made a series of observations, both on the prophylaxis and treatment of scarlatina by means of sodium sulphocarbolate. He has promised this evening to give us the results of his extensive experience. As a prophylactic in conjunction with isolation, I do not remember of having had second cases in the same family where use was made of this drug; but without isolation it failed in my hands, possibly because the drug was not used in sufficiently large doses, in consequence of the sickness and diarrhœa which sometimes followed its use; but in those cases which occurred after the sulphocarbolate had been used as a prophylactic, I believed the disease to be milder. In the treatment of scarlatina for some years I have in all cases used the *carbonate of ammonia*, given in small doses at short intervals. This treatment was, I think, introduced by Peart. It has been used by others in severe cases, and by some with such results that they look upon it as almost a specific. I can hardly claim this for it, but have got better results than by other methods. Its mode of action I cannot explain, but it has the advantage of being a stimulant to the nervous system, which is profoundly affected in severe cases. It is further useful by acting on the skin, which in this disease is so dry. The objection to it is its tendency in some patients to produce gastro-intestinal irritation, except in very small doses. *High temperature* is a symptom which occasionally calls for treatment. Antipyrine and kairine are so liable to be followed by prostration that I have never used them. Antifebrin I would have no fear of, but have not tried it. My experience accords

with that of Dr Jamieson as to the use of the bath, but the wet pack is also in some cases a most important adjunct. I well remember one of the first cases in which I used it—a child with constant convulsions in consequence of high temperature at the commencement of scarlatina. By means of the bath the temperature was lowered and the convulsions ceased, but on each occasion, shortly after removal from the bath, the temperature rose and the convulsions returned. In this and another case the pack was required for two days without intermission; its ease of application and the relief it afforded were striking. Another of the earlier difficulties is the *severe form of sore-throat* which sometimes sets in about the fifth or sixth day of the fever. Children with enlarged tonsils are, in my experience, more liable to it than others. Its cause I believe to be zymotic, but not the same organism as that of diphtheria. That disease may supervene upon scarlatina as it may upon measles or other debilitating illness, the patient being then susceptible to infection by the diphtheritic poison. I have seen only one case of diphtheria during convalescence from scarlatina, and believe that the sore throat now referred to is clearly differentiated by the absence of true false membrane, by its mode of extension, and the absence of paralytic sequelæ. Believing the throat to be in this disease "the feeding ground of micro-organisms," as Dr Hunter at our last meeting aptly put it, which micro-organisms afterwards affect the system in various ways, I have for some years insisted upon the use of antiseptic applications and gargles in all cases of scarlatina from the commencement of the disease. Since the adoption of this course I have seen fewer cases of severe throat and nose affection than formerly, and fewer cases of inflammation of the glands and connective tissue of the neck. When this severe throat, nose, and ear affection does supervene, what treatment should be adopted? My own practice has been to use locally antiseptic applications, sprays, and gargles, at short intervals, and internally chlorate of potash and iron in mixture. Occasionally we have in the course of scarlatina *elevations of temperature* without evidence of inflammatory complications, and no history of error in diet to account for the symptoms. After the tongue has become moist, the swallowing more easy, the fever has abated, and the patient rests quietly at night, there is a recurrence of all the symptoms of the first few days of the fever. Are not these *relapses* analogous to those we meet with in typhoid fever? That second attacks do occur later on during convalescence has been clearly established, the patient passing through a second illness with a second desquamation. *Dieting* of some patients constitutes a difficulty calling for great care both as to quantity and to quality of food, any error in these respects setting up the temperature even to a considerable extent. Rich soups and animal food should not be given till convalescence is well advanced. *Rheumatic complications* are in my experience more common after severe sore-throat.

In the treatment of them salicylic acid and salicylate of soda should, I believe, be avoided, as these remedies are excreted by the kidneys. Perhaps the most frequent complication is *nephritis*. Are we in a position to indicate the reason of its occurrence in some cases, its absence in others? Some think it is more frequent when the poison is introduced by means of milk, and that milk epidemics are more severe. I am not satisfied that it is more common in milk epidemics. Post-scarlatinal nephritis is certainly not by any means uncommon after the mildest types of scarlatina, and it is often absent after the most severe. It does occur even when from the beginning of the disease the greatest care has been taken both as regards dietary and avoidance of chills. As a predisposing cause of post-scarlatinal nephritis must we not recognise a structural weakness of the kidney? The main exciting causes are chills to the surface of the body, specially during the first fortnight of desquamation, errors in diet, and medicines which are irritating to the kidney. Until the desquamation is almost entirely completed, while the new surface is yet tender, the patient should be confined to bed. Animal food should not be given till well on in convalescence, and if there be any trace of albuminuria it should be withheld for a longer time. In *uræmic convulsions* my experience leads me to prefer subcutaneous injections of pilocarpine to any other method. Whatever be the antiseptic application to the skin, infection remains, according to my observation, until desquamation is entirely completed. Perhaps before this the question might have been raised, Whether, as scarlatina is a self-limiting disease, we should not simply place the patient in favourable hygienic conditions, give directions as to feeding, and with a kindly word, a placid smile, and folded hands, stand by and watch the natural course of the disease? If the case be an extremely mild one, we may afford to do this, but in dealing with so treacherous an affection, as our President has often characterized it, we have always to be on the look-out for complications; and in the more severe cases we can, I believe, by judicious treatment, beneficially modify the course of the disease as well as prevent some of the complications.

Dr James Carmichael, with reference to the remarks made at the last meeting of the Society by several members in regard to cows' milk as a vehicle for diffusion of the scarlatinal poison, particularly in the recent epidemic which occurred in Edinburgh, said that it appeared to him that no *proof* had been brought forward that the epidemic had originated from this source. The disease was generally diffused all over the city, but specially prevalent in the western district. Now, it appeared that this was the rule in such epidemics, certain localities being specially attacked and suffering more than others. Certain local causes were often discovered to account for this, and so it was in this epidemic—the sanitary conditions generally, and particularly the drainage, being

so defective in part of the district referred to, that the authorities thought it necessary to have all the drains renewed. One case of scarlatina had been found to exist in a farm-house, the dairy of which supplied part of the West End of the city; but it had not been proved, so far as the Health Committee's reports indicated, that the milk had been contaminated directly or indirectly. No evidence had been given to show that any communication by persons or fomites had taken place between the patient and the dairy. It had not been demonstrated that particles of desquamated skin got into the milk, or that in any way it had been infected. He admitted that cows' milk was a most suitable medium for conveying such a poison as scarlatina. It was a septic fluid, and one in which the organisms found to exist in scarlatinal tissue multiplied with great rapidity. It had been proved in other epidemics, notably in one which occurred many years ago in a large town in a neighbouring county, that an epidemic had actually originated from contamination of the milk with desquamating cuticle from a milkmaid who was ill with the disease. This proof he accepted as one quite convincing and of real scientific value; but in the recent epidemic no such evidence existed; and he thought, as medical men, they could not accept as proof that the milk was poisoned the fact that a patient in the neighbouring farm-house suffered from the disease. No doubt the existence of this case was sufficient to arouse suspicions of poisoning, but no evidence had been brought forward to prove direct contamination of the milk. In the short time at his disposal he desired to confine his remarks chiefly to the question of treatment. In regard to prophylaxis, three methods deserved notice —isolation, inoculation, and disinfection. There could be no question about the desirability in all cases of isolation, and on this the profession were unanimous. Various drugs had been used by administration to patients in infected areas. The first remedy which had been proposed for this purpose was belladonna. He had tried it frequently in the earlier years of his practice with entirely negative results, and this, he thought, had been the general experience of the profession. Antiseptic remedies, and particularly the sulphocarbolate of sodium, were largely used, and it appeared to him with encouraging results. The drug should be given in full doses and for several weeks, being taken along with food. Boracic acid and listerine had been employed for the same purpose. The next method of prophylaxis was the ideal one which many physicians were seeking to realize, viz., inoculation. Dr Strickler, in America, was one of the first to put this method on its trial by injecting into children septic material from a horse supposed to be suffering from the disease. The children contracted local sores at the point of inoculation, and in many cases lymphatic glandular enlargement in the vicinity resulted, and subsequent immunity from the disease on exposure to infection. Let them hope that the time might

not be far distant when inoculation with material from a similar or modified disease in the lower animals, or an attenuated virus prepared for the purpose, might be offered to the public as a hopeful means of arresting the progress of this scourge. *Disinfection* was now invariably used as a ready and efficacious means of preventing the spread of the disease. This could be most suitably and successfully carried out by keeping the room well ventilated, and by disinfecting the body-clothes and excreta of the patient as well as the patient himself. Too much importance could not be attached to thorough ventilation of the room, which should contain no articles of furniture except those absolutely necessary; bed curtains should be removed and carpets taken up. The room should never be swept, but instead, the floor mopped over every day with water containing 15 per cent. of a solution[1] of carbolic acid. This he considered a precaution of the greatest value. In directing attention to the patient himself, all cast-off articles of clothing should be thoroughly disinfected by steeping for twenty-four hours in a solution of carbolic acid in water, and then boiling or steaming. After the febrile stage was over, there were two methods of disinfection, by medicated baths and unguents applied to the cutaneous surface. He had no hesitation in giving a preference to baths; they were soothing to the patient, and assisted materially the desquamation. He was in the habit of giving them every other day during the process of peeling. Carbolic acid or mercuric chloride were the most efficacious disinfectants. Under this treatment the skin over the body and limbs readily desquamated. The palms of the hands and soles of the feet were best treated with an ointment which he had already shown to the Society, composed of salicylic acid, ol. of eucalyptus, with lanolin as a basis. The application of oily preparations and ointments to the entire cutaneous surface he had entirely discarded in hospital practice, reserving their use occasionally to those cases in private practice where there appeared to be some special advantage or reason for their application. He considered them neither so cleanly nor efficacious in assisting desquamation as the treatment by baths alone. His friend Dr Jamieson in his valuable paper attached the greatest importance to the disinfection of the skin, particularly by unguents and oily applications, and claimed for it, if properly carried out, specific properties as a perfect method of prophylaxis. He detailed a case in which one member of a family attacked by the disease passed through it favourably, and none of the other children, although isolation was not carried out, were affected with the disease. Formerly, before any such methods of prevention were in vogue, it was not by any means an uncommon thing to find only one, or perhaps two members, of a family, say of five or six, attacked with scarlatina, and the others escape, even although

[1] The nurse should be provided with a solution of carbolic acid in water 1-15, a pint of this being added to a gallon of water.

isolation of the patient had not been resorted to. This seemed to weaken Dr Jamieson's argument in favour of his method of inunction. The truth was, the scarlatinal poison, although one of the most persistent known, was not so readily diffusible or volatile as that of measles, typhus, or small-pox, which probably accounted for the fact that it did not always spread in families with such readiness as was generally supposed. The cardinal rules in the further treatment of cases of scarlatina were keeping the patient in bed for at least three weeks during the greater part of the desquamating period, administering during this time chiefly milk and farinaceous diet, with light soups, animal food not being given in ordinary cases till the end of the fifth or sixth week. Under such treatment his experience, both in hospital and private practice, was, that the occurrence of complications, especially of the kidney, was comparatively rare. He should like to say a word regarding the use of antipyretics. He had used quinine, antipyrine, and antifebrine with frequent good results. He seldom had resorted to such remedies unless the temperature persisted above 103°. Quinine, antipyrine, and less frequently antifebrine, sometimes produced unpleasant symptoms, especially sickness or depression, and had to be discontinued. He considered, of all the antipyretic remedies, cold affusion, sponging, or wet pack were the most grateful, the safest, and most efficacious. Sponging he generally tried first, and if the temperature was not sufficiently lowered the other methods were employed. He used few drugs in ordinary uncomplicated cases, especially in hospital practice. In private patients it was customary to order diaphoretic salines or other simple remedies. In regard to throat applications, these varied according to the local conditions. In simple catarrhal or slight follicular affections applications were not always necessary. In ulceration or diphtheritic complications he had found diluted carbolic glycerine, lactic acid, and undiluted Condy's fluid very useful, or liq. ferri perchlor. in glycerine, with ½ gr. to the ounce of hydrarg. perchlor. He should like here to emphasize the fact which his experience had borne out, that in a small proportion of cases of well-marked, some of them severe cases, no throat affection was noted, not even the slightest catarrhal redness, showing that throat inflammation, although generally, was not invariably present in scarlatina.

Dr P. A. Young said there were many points of importance in a discussion on scarlatina which were not yet removed from the domain of theory, but that the research which gave rise to this discussion would do a good deal to give some data from which to start. The cases which were of terrible interest to the practitioner were the malignant ones. What was the causation of such cases? They often found more than one case in the same family. Did the bacilli enter the body in overwhelming numbers, or did they increase with great rapidity? It appears from several considera-

tions that the bacilli did not of themselves cause the disease, but that acting on some suitable pabulum in the blood they give rise to a poison which was the true scarlatina poison. In zymotic disease it was usually considered that three factors existed—one outside the body; secondly, a pabulum in the blood; and thirdly, a poison which was the result of the first factor acting on the second. The observations of Drs Jamieson and Edington would lead them to believe that the factor outside the body in scarlatina was the bacillus scarlatinæ. What part of the blood was acted upon had yet to be studied, and in order to get the resulting poison, the aid of the chemist must be called in. In Sir Henry Roscoe's inaugural paper before the British Association this subject was very suggestively brought forward. From the alkaline condition of the blood in many fevers, it was probable that this poison was of an alkaline nature. The symptoms observed in malignant cases were very like those seen in typhus—the muttering delirium, the dusky rash showing that the poison was acting on the vaso-motor centres, and the high temperature. It had been observed that there was often some family tendency to malignancy, and frequently it was found that insanitary conditions of the dwellings led to a development of these bad cases. So that it appeared probable that sewer poison was often a cause of a case showing malignant symptoms. As regards treatment, wet packing had been tried, as well as a variety of antipyretics, but, as a rule, a malignant case proved fatal in spite of all treatment. He remembered that when he was a student, Professor Maclagan had told them the astonishment that had been created by his having a second attack of measles, and that his father, who was a medical man, had brought his friends to see this phenomenon. Now-a-days second and third attacks of measles and scarlatina were common. What was the cause of this? It could not now be said, as they have formerly taught, that one attack prevented a second. For the sore-throat of scarlatina it was important to pay attention to the local treatment in order to prevent glandular enlargement and suppuration with its disfiguring consequences. When there was middle-ear suppuration it had been recommended to open the drum head. This must be a difficult operation in a child suffering from fever, and when the canal of the external meatus was so often contracted by inflammatory swelling. He would like to hear the opinions of specialists present on the subject. Kidney complications were usually produced by cold or errors of diet. The best preventives were, bed for a month and milk diet. To prevent the desquamation being infectious he formerly employed camphorated oil, but since antiseptics had come into use he applied inunctions of carbolic oil.

Dr Illingworth said it was not necessary for him to advocate the use of germicidal remedies in the treatment of scarlet fever. Such treatment was admitted to be based upon sound rational

principles. To the throat affection he was of opinion they should direct particular attention, it being the source of the continued infection of the blood by absorption of infective material lodged and developed there. In those cases where no throat affection had been observed, he would suggest that it had either disappeared before the case came under observation, or had been more pharyngeal or naso-pharyngeal than tonsillar, and had thus escaped detection. Treatment by the biniodide of mercury he had found of immense service in modifying the course of scarlet fever, reducing the temperature, checking or altogether preventing the inflammation of the skin, and preventing the dreaded sequelæ. These results it would appear to effect by its powerful germicidal properties, which, according to well-known investigators, were unrivalled. He first prescribed the drug for symptoms of meningitis in a case of scarlet fever, and it gave such good results that he gave it in others, and had the satisfaction of observing that it had a marked effect upon the disease in all. In some the rash disappeared on the second day, reappearing on discontinuing the medicine, again to disappear on returning to it again. In all there was a more rapid recovery than with other means of treatment he had adopted. For a child of about seven years he gave half-drachm doses of the bichloride of mercury solution, with one and a half or two-grain doses of the iodide of potassium every two, three, or four hours. The biniodide of mercury was thus held in solution in a large excess of iodide of potassium, and mercurialism was thus prevented. For younger children half those doses would be sufficient. It might be that a less dose of the iodide of potassium would answer equally well. If there were a dry tongue and other urgent febrile symptoms, the acetate of ammonia or the salicylate of soda might be added; but he strongly advocated the administration of iron as soon as ever the temperature had become normal and the rash had disappeared. Dr Ringwood of Kells informed him that he prescribed the biniodide and iron every two hours alternately from the first onset of the disease, and that he had most gratifying results. With regard to local treatment, it was only quite recently that he had used the biniodide of mercury to the throat. From the five or six cases in which he had tried its effect he had had excellent results. His plan was to precipitate the salt from two ounces of the bichloride of mercury solution with about one grain of the iodide of sodium or potassium. To this he added a little glycerine to keep the particles in suspension, and rapidly but thoroughly applied it twice a day with a laryngeal brush.[1] The material being sweet, children did not much object to it, and the parents or nurse could easily be instructed to apply it oftener if desirable.

[1] The exact formula is the following :—Add 10 minims of a 1 in 4 solution of potassic iodide to ℥iss of a 1 in 500 solution of the bichloride, and sweeten with glycerine.

In malignant scarlet fever they had, he thought, to deal with a condition of the blood similar to that which was present in all cases of this disease and other exanthemata sooner or later in their progress—a condition of over-fluidity, due, he believed, to the depredations of micro-organisms upon the hæmoglobin and fibrin elements of the corpuscles. The only difference was that in the malignant cases it was more rapidly developed from the intensity of the poison, and collapse was induced, as indicated by the dusky hue of the face and skin generally, scanty rash, prostration of vital energy, and very frequently diarrhœa. With those who had already spoken, he must admit that he had had many fatal cases of this kind, but he had saved several by giving iron. Iron was, he believed, the only remedy which could supply those fibrin elements which had been lost, and it should be given in full doses every half-hour or hour. Antipyretics he had never used, and he was careful to avoid, firstly, because the giving of them was in a sense treating symptoms only; and, secondly, because some, such as antipyrine and antifebrine, have been known themselves to produce cyanosis and other dangerous conditions. As for diet, he had found milk of great service when given at regular intervals of three or four hours, so as to allow of due digestion and assimilation. In scarlatinal dropsy he had had the best results with iron and occasional doses of the compound jalap powder. In convulsions from this cause he had saved life by venesection to three, four, six, and even nine ounces in young children. The chief source of infection he believed to be the throat. He had no hesitation in placing children with others at the end of the tenth or twelfth day, provided their throats were perfectly free from inflammatory and ulcerative mischief, whether there was desquamation of the cuticle or not. He also thought that one, or at the outside two carbolic soap baths about the tenth day were quite sufficient to prevent infection from the skin. He should especially here refer to those cases of scarlet fever in which there was such an irritable state of the stomach that the biniodide mixture was continually vomited. In these he had found a combination of bismuth and hydrocyanic acid to answer well.[1]

Dr Buist said that in regard to the etiology of infective diseases bacteriologists were surrounded by considerable difficulty. It was by no means easy to say in what locality the infective material was best found and what form it took. Although they knew that the infective material of scarlet fever existed in the skin, perhaps in the blood, and also in the secretions of the throat, they had not yet determined the form in which this contagium existed in these situations, and that was of importance when they came to cultivate. The researches of Klein and Edington were defective in this

[1] The biniodide in such cases could be given in powders of $\frac{1}{16}$th grain with powdered sugar three times a day.

respect. Neither of them had defined the form of the bacterium present in the natural materials. But when they came to pure cultivations of the materials the matter changed, whether they obtained cultivations of streptococci or of bacilli. When they came to inquire into the physiological action of cultivations, they found a difference of opinion with regard to the effect of the streptococcus and with regard to the effect of the bacillus. Which of the organisms was specific? Was either or neither? These were questions he did not profess to determine. They arose from what had been placed before them. He thought this was a link wanting in the chain of evidence. They had no information as to the bacteric form existing in the natural infective materials, and the observers were at variance as to the action of their pure cultivations. With regard to their experimental work, he thought that in both researches it was wanting. That of Klein had been carried out both by inoculation and by feeding, and it had this advantage, that an accidental transmission of the disease had been made to the human subject without, however, onset of a scarlet rash. There was merely a vesiculation or ulceration. Was this what they usually understood by scarlet fever? In Edington's experimental work, so far as it went, he thought that the bacillus appeared to produce a scarlet eruption on the skin because on various occasions he produced such an eruption on calves and other animals when inoculation was performed; but in the case of one calf feeding and inoculation were followed by death in a very few hours, and this showed the difference of effect according to the difference in the mode of introduction of the material. He believed that the moribund calf had shown a very vivid scarlet rash, an important fact which was omitted from his paper by Mr Edington, and this omission greatly detracted from its value. From the spleen of this dead calf Mr Edington succeeded in cultivating his bacillus. It was a pity that he had not inoculated another calf with this cultivation. The other organisms which Mr Edington described as concomitants, he thought must be regarded as non-specific or non-pathogenic. They might be common to the normal skin. With regard to the form in which the contagium existed in the skin, Dr Allan Jamieson said he thought it existed in the flakes in the mature form. If so, and if the bacillus were the true specific cause of scarlet fever, they should find it easily demonstrated, whereas in Mr Edington's paper it was stated that only minute points could be detected which he considered as spores, and he believed that his bacillus developed from these. He thought that this observation showed that the contagium existed in the flakes of skin in the embryonic and not in the mature condition. He should like that in future investigations the tissues and the secretions from the throat should be examined, because from them as well as from the skin and blood were they likely to get the specific organism of the disease.

Dr Murdoch Brown asked if Dr Carmichael, believing in isolation, considered himself justified in allowing healthy children to mingle with the infected. He also wished to know if Dr Carmichael would permit his own household to consume milk coming from a dairy where a case of scarlet fever was known to be. As to the disinfection of the chamber by sulphurous acid, was this fumigation carried on during the illness of the patient or after? He was of opinion that the 15 per cent. solution of carbolic acid would prove detrimental to the integuments of those with whom it came in contact. He wished to know from Dr Edington whether his brother's blood had been examined before the milk containing the cultivation of streptococci had been administered?

Dr James Carmichael replied that the solution of carbolic acid referred to was intended for the floor and articles of furniture, not for the skin, as Dr Murdoch Brown supposed. He used the sulphurous acid both during the progress of the case and to disinfect the room afterwards. In the case of the family referred to, it was not possible to carry out isolation of the patient.

Dr Edington said his brother's blood had been examined, and was not found to contain any organisms before the administration of the cultivation of streptococci. They all disappeared from the blood in about three weeks after. He did not think isolation of these organisms was so easy as some seemed to suppose. As a matter of fact, plate cultivations were rather difficult. An impure test-tube cultivation might mean one hundred cultivations before purity could be obtained.

Dr Allan Jamieson said he was glad Dr Carmichael had explained away any danger of suffocation through his fumigations as Dr Murdoch Brown had suggested, but he thought the plan of swabbing the floor might increase the risk of rheumatism, one of the most common of the complications of scarlet fever. In his remarks on prophylaxis, Dr Illingworth had said nothing of the biniodide of mercury which he had recommended to be taken by those in attendance on the patients for that purpose. The other day a lady had shown him a prescription which she had taken to ward off an attack in her own person, and asked if it could be the cause of swelling of the eyes, and an outbreak of spots from which she had suffered since commencing it. The prescription contained in each dose one-sixteenth of a grain of perchloride of mercury, and three grains of iodide of potass in tincture of cinchona, to be taken thrice a day for an indefinite period. It had been sent by her mother in England, who had got it from the family doctor, with the assurance that it would prove a certain prophylactic. Some caution surely ought to be exercised in recommending such a powerful remedy. He was surprised to hear it stated that desquamation did not ensue when the biniodide was employed in the treatment of scarlet fever, as it was invariable under all other modes of management. Dr Ritchie had asked for experience

of the salicyl compounds. Salicin, but not the salicylates, nor salicylic acid had been used very freely in the City Hospital in instances of rheumatic complications, and in not one of the cases in which it had been given did any nephritic symptoms manifest themselves. As to the condition of the organism in the scales, he did not mean that it was mature morphologically, but that it ripened in these and was cast off in a state ready for immediate reproduction so soon as it was placed in a suitable medium. He thought that a strong argument in favour of the baths and inunctions which he had recommended and practised was, that whenever they were so used the spread of the disease to other and unprotected members of the household was invariably prevented even when strict isolation could not be carried out.

Dr Illingworth asked leave to reply to Dr Jamieson. In recommending the use of the bichloride of mercury with iodide of potassium as a prophylactic for scarlet fever, he never advised so large a dose as five grains of the iodide three times a day. It would be sufficient to induce iodism, or even an iodide rash. But a thirty-second of a grain of the perchloride of mercury, with two grains of the iodide, might be taken for months without any untoward effect. In regard to desquamation, he did not affirm that by the biniodide treatment it was always prevented. He had found it less marked than with other treatment, and in some cases altogether prevented. It must be noted, also, that it was not by him alone that this had been found to be the case. Dr Clement Dukes stated that the physician came to doubt whether the case had been one of scarlet fever or not, so rapid was the effect upon the skin in many cases. This effect, he believed, would be still more marked when the throat was thoroughly and frequently treated with the biniodide for the purpose of rooting out the initial lesion. Mr Hoadley Gabb, of Hastings, wrote him to the effect that being called in to a case of undoubted scarlet fever with a temperature of 102°, he gave the biniodide, with the effect that in the course of twenty-four hours the temperature was reduced to the normal, and the rash disappeared. The family doctor then called, and, in the absence of rash, pronounced it a case of roetheln. The parents being dissatisfied, called in another consulting physician, who avowed his inability to decide between his brethren until he had waited for desquamation. The amusing part of the story lay in the fact that he was still waiting for that process. He agreed with Dr Buist that the throat had not had the attention it deserved. When he found large numbers of micro-organisms in the exudation from the throat in 1886, they all seemed to him to have the same characters, so that he thought there would not be much difficulty in isolating the chief ones. Further, the scarlatinal organism must have, he thought, such intense power, that from that reason alone it would preclude the possibility of others growing readily in its vicinity.

Mr Cathcart asked if any of the investigators had found an organism like the tubercle bacillus in the blood in their scarlatinal researches, as a strumous or tubercular taint was so apt to be a sequela of scarlet fever.

Dr Edington remarked that he had examined six post-scarlatinal abscesses, and in none of them found either tubercle bacilli or any of the organisms described in connexion with that disease.

Mr Hare said it was generally admitted that it was the rarest thing to get the tubercle bacillus in the blood. It was conveyed by the lymphatics, sometimes by the perivascular lymphatics, but very rarely by the blood channels themselves.

The President, in closing the discussion, said that he felt disappointed that the discussion had elicited so little of importance, either on the scientific or on the practical side of the question. The committee's report was of course, in his opinion, of some value, as affording a measure of confirmation of Mr Edington's work, and it was scarcely to be expected that much additional information could at the present stage have been offered by individual members. But as to the treatment, he had hoped for a much more valuable discussion. He had hoped that some light would be thrown on the treatment of the severer cases they had to deal with, and he was certainly indebted to the gentlemen who had given the results of their experience; but it was clear that little definite had been elicited. As to the question of prophylaxis, also, they had learned but little. They did not know whether the sulpho-carbolate of soda had been found by others to have the good effects claimed for it by Dr Brakenridge. As to the biniodide, they were glad to have heard from Dr Illingworth's own lips some of the experience he had already given to the profession on the subject. His contention was still *sub judice*, however, and by no means settled. The profession was still feeling about for some sure method of prophylaxis. He thought that something had been gained as to the management of the throat affection. Every one had been alive to it more or less, but this discussion would tend to increase the sense of its importance. All must feel they had reason to be humble in regard to their power in dealing with fever. Their art was still imperfect, and they must work hard to obtain better means of meeting the difficulties which met them all at one time or another.

INDEX.

ERRATUM.

Page 183, line 8, *for* "Marcy's polygraph" *read* "Marey's polygraph."

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LAWS

OF THE

MEDICO-CHIRURGICAL SOCIETY

OF

EDINBURGH.

INSTITUTED 1821.

Printed for the Society

BY

OLIVER AND BOYD, EDINBURGH.

1888.

CONTENTS.

LAWS.

I.—NAME AND OBJECTS OF THE SOCIETY.

1. The Society shall be denominated The Medico-Chirurgical Society of Edinburgh.

2. The Objects of the Society shall be—To receive communications on Medicine and Surgery, and subjects connected therewith; to converse on medical topics; and to promote professional improvement by any other means that may, from time to time, be approved by the Society.

II.—CONSTITUTION OF THE SOCIETY.

The Society shall consist of Ordinary, Corresponding, and Honorary Members.

1. Of Ordinary Members.

1. Ordinary Members must be legally qualified medical practitioners.

2. Any person proposed as a Member must be recommended by two of the Ordinary Members of the Society. His name and qualifications must be intimated to the Secretary, at least fourteen days before the meeting at which he is to be balloted for. Applications shall be intimated in the billet for the Ordinary Meeting thereafter, and then disposed of by ballot; and no person shall be declared elected unless three-fourths of the votes appear in his favour.

3. Every applicant for membership shall be required to lodge with the Treasurer the entry-money of Two Guineas, previous to his name being proposed to the Society for admission, and

this sum shall be retained by the Society in the event of his election.

4. The Ordinary Members, both Resident and Non-Resident, shall have equal privileges, and shall pay to the Treasurer of the Society a sum not exceeding One Guinea annually; but the Non-Resident Members who do not desire to vote, or otherwise take part in the private business of the Society, shall be excused from paying the annual subscription.

5. If a Non-Resident Member who has never paid, or who has ceased to pay the annual subscription, and to take part in the private business of the Society as aforesaid, should at any time desire to be placed on the Roll of Members entitled to vote, he shall give intimation of such desire to the Secretary, and declare his willingness to become liable for the annual subscription. And it shall be competent for the Secretary, with the concurrence of the Council, to place the name of such Member on the Roll of Members entitled to vote, within three months of the date of his application.

2. Of Honorary Members.

1. The Honorary Members shall be gentlemen of distinguished attainments in Medicine or the allied sciences, not being Ordinary Resident Members of the Society. Their number shall be limited to Ten.

2. If an Ordinary Non-Resident Member be elected an Honorary Member, he shall, *ipso facto*, cease to be an Ordinary Member.

3. At the first meeting in March of each session, the vacancies in the list of Honorary Members shall be announced by the President, who shall on the same occasion invite the Members present to nominate fit and proper persons to supply the vacancies. The nomination list shall remain with the Secretaries for a fortnight thereafter for the admission of new

names. The Council shall then meet, and in the event of there being more than three nominations for each vacancy, the Council shall frame a list of three for each vacancy. This list shall be printed in the billets issued for the first Meeting in April.

4. The voting upon the lists framed as above shall be taken at the first Meeting in April, the lists of candidates nominated, as finally arranged, being announced by the President. Each Member present shall be entitled to vote for one name in the case of each vacancy; and the persons having the greatest number of votes so taken shall be declared to have the majority of votes of the Society, and to be appointed Honorary Members.

5. Every person elected an Honorary Member shall have immediate notice sent him by the Secretary, along with a Diploma of the Society.

3. Of Corresponding Members.

1. The Corresponding Members shall be gentlemen distinguished in Medical Science, not Ordinary Resident Members of the Society.

2. If an Ordinary Non-Resident Member be elected a Corresponding Member, he shall, *ipso facto*, cease to be an Ordinary Member.

3. At the first Meeting in March of each Session, any four Ordinary Members may propose in writing the name of any individual whom they deem suitable for the Diploma of Corresponding Member. The names proposed shall be submitted to the Council, and, if approved of, shall be printed in the billets for the first Meeting in April, and be then balloted for. No person shall be declared elected unless two-thirds of the votes appear in his favour.

4. Every person elected a Corresponding Member shall

have immediate notice sent him by the Secretary, along with a Diploma of the Society.

4. Diplomas.

The Society shall grant to Honorary and Corresponding Members a Diploma to the following effect:—"*Societas Medico-Chirurgica Edinburgena, anno salutis* 1821 *instituta, omnibus ad quos hæc pervenerint salutem. Eximium ornatissimumque virum. . . . . quem scientiam medicam ingenio felici et laudabili diligentiâ coluisse compertum habemus, in numerum Sociorum* (externorum, honorariorum) *adscripsimus. In cujus rei fidem, has literas, manu præsidis nostri subscriptas, emitti lubenter jussimus.*

..... *Præses.*

..... } *a Secretis.*
..... }

"*Edinburgi Anno Domini Dic.*"

III.—OFFICE-BEARERS AND COUNCIL.

1. None but Ordinary Resident Members shall be entitled to hold offices in the Society.

2. The Office-Bearers shall consist of a President, three Vice-Presidents, two Secretaries, a Treasurer, and an Editor of Transactions, who, together with eight other Members, shall constitute the Council.

3. The election of the Office-Bearers shall take place by ballot, at the first Meeting in November.

Council. 4. Five Members of the Council shall constitute a quorum.

5. The Council shall regulate the private business of the Society. But it shall be in the power of six Ordinary Members, concurring, to bring any act of the Council under the review of the Society, provided they intimate their intention within one month from the date of the act.

6. Four Members of the Council, exclusive of the Office-Bearers, shall go out annually, and shall not be again eligible for a year.

7. The President shall continue in office for two years. President.

8. One of the Vice-Presidents shall go out of office annually in rotation. Vice-Presidents.

9. The Secretaries, Treasurer, and Editor of Transactions shall be elected annually. Secretaries.

10. The *Secretaries* shall take minutes of the meetings of the Society, and enter them in the minute-book, which shall be open to the inspection of the Members. They shall also have the management of the correspondence of the Society. The minutes of the Society shall report the business, both public and private, of each ordinary meeting.

11. The funds of the Society shall be deposited in one of the chartered banks of the city, in the name of the Treasurer and senior Secretary of the Society, in their official capacity. The Treasurer shall not retain in his hands more than £10 without the sanction of the Council, and he shall not be entitled to draw any sum without the concurrent signature of the Secretary. Treasurer.

12. A statement of the funds shall be presented to the Society by the Treasurer at the first meeting in November of each year. At that meeting the Society shall appoint a Committee to audit the accounts; and the financial statement, duly vouched, shall be printed and circulated among

the Members along with the billet for the first meeting of the Society in December.

13. The Office-Bearers, on going out of office, shall immediately be re-eligible, with the exception specified, chap. iii. § 6.

14. All motions relating to new laws, or to alterations on existing laws, shall be made in writing at one meeting, printed in the billets for, and considered at the following meeting. The concurrence of two-thirds of the meeting shall be required in order that any such motion may become law.

IV.—MEETINGS OF THE SOCIETY.

1. Each Session of the Society shall commence on the first Wednesday of November, and shall continue for nine months; the Meetings to be held on the first Wednesday of each month, at 8 P.M., except when otherwise ordered by the Council.

2. Additional Meetings may be held when the Council think it necessary.

3. At Ordinary meetings five shall be a quorum; but twelve shall be necessary for the election of Members.

4. Members shall, as far as possible, give due notice to the Secretaries, at least Ten days previous to the Meeting, of such Patients, Pathological Specimens, etc., as they may propose to exhibit, so that a list of these may be included in the billets.

V.—VISITORS.

1. Each Member shall have the privilege of introducing one Visitor at each Ordinary Meeting.

2. Visitors may take part in the Discussion if individually called upon by the President, and are to retire at the commencement of Private Business.